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ORIGINAL MEMOIRS

THE PRESENT STATUS OF BISMUTH PASTE TREATMENT OF SUPPURATIVE SINUSES AND EMPYEMA.*

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NEARLY eight years have now elapsed since I began to employ the bismuth in diagnosis and treatment in sinuses, etc. During this period a variety of cases amounting now to over 1100 cases have been treated by myself and my brothers, which naturally have given us ample opportunity to give the method a fair test. The experience gained from successes and also failures has taught us valuable points, which I shall now bring before you, in order that they may aid you also in correctly applying this method.

Many of you have no doubt had experience of your own in treating abscesses and sinuses with bismuth paste, and are familiar with the method from the literature. This relieves me in a degree of the necessity of discussing the theoretical side of the subject.

In June, 1906, I demonstrated before the Chicago Medical Society this new method, which consists in injecting the sinuses with a mixture of 33 per cent. of bismuth subnitrate and 66 per cent. of vaseline. This mixture must be sterile and liquefied by heating, so that with moderate pressure it

* Read before the International Medical Congress, London, 1913.

will fill all branches of the sinuses. Radiograms taken of the injected region show in perfect clearness the extent and ramifications of the fistulous tracts, and often lead us to the focus of disease (Fig. 1). A glance at such picture enables us to discriminate between operable and inoperable cases, whereas with the older diagnostic aids, such as the probe or the injection of colored fluids, the operation itself had to be performed in order to determine whether the case was operable or not.

This diagnostic method led to the discovery that the injection of the bismuth paste has a distinct therapeutic effect. This was not fully appreciated until a year later when we observed that the patients on whom we had employed the injection for diagnostic purposes returned to us after months entirely cured. This at once suggested the use of bismuth-vaseline paste for curative purposes, and our expectations were far surpassed when we tried it in a number of obstinate cases.

On January 15, 1908, I brought before the Chicago Medical Society the first fourteen cases treated by this method, ten of which were then cured. Of these fourteen cases, thirteen are now entirely healed, one died in 1910 after sixteen years, suffering with most extensive necrosis of the spinal column.

Soon after my first publication in the *Journal of the American Medical Association* and the *Centralblatt fur Chirurgie*, surgeons in all parts of the world began to employ the bismuth paste. Their readiness to give it a trial was partly due to the simplicity of the method applicable to a class of cases for which there was no efficient remedy. Suitable cases for a trial were abundant everywhere, and only too willing to try anything new.

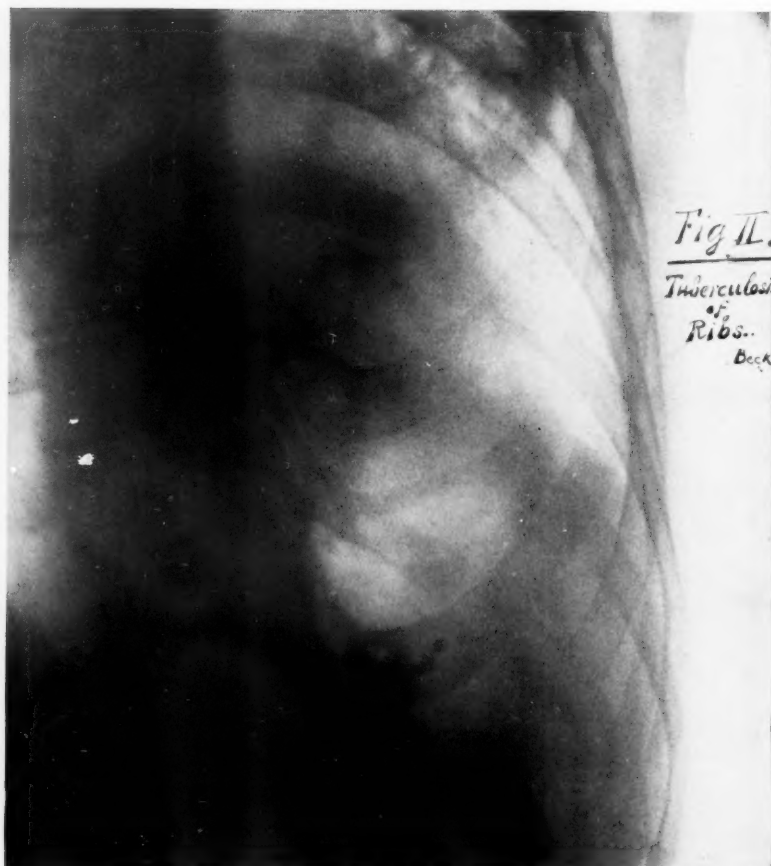
Reports soon began to find their way into literature. Some authors obtained results even better than ours, others were only partially successful, and then in the hands of a few the method was a failure. Considering, however, that 100 per cent. of these cases in which it was tried had already been treated by other methods without success, we must regard

FIG. 1.



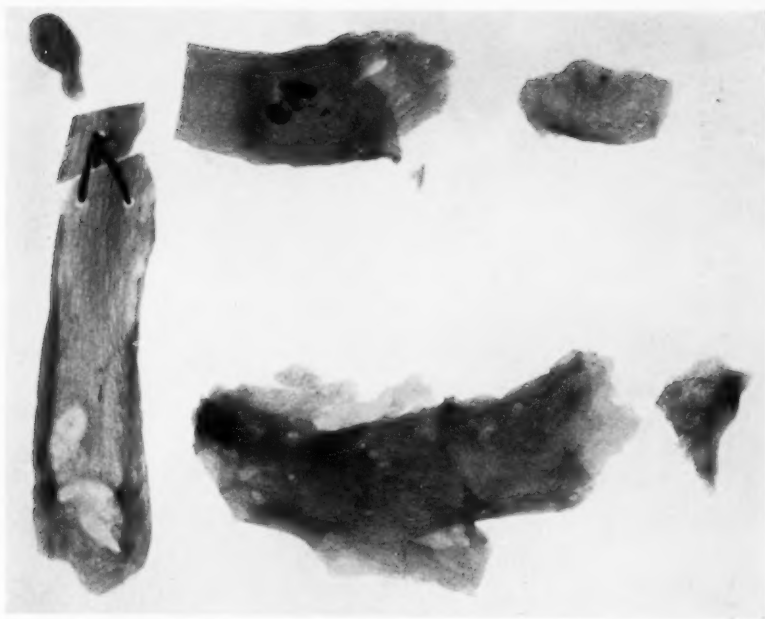
Network of sinuses originating from hip-joint.

FIG. 2.



Tuberculosis of seventh and eighth ribs. Bismuth injected through sinus in sternum.

FIG. 3.



Resected ribs showing that disease existed within the cancellous tissue.

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SINUS PASSING THROUGH SPINAL COLUMN,
INJECTED WITH BISMUTH PASTE.

Beck.

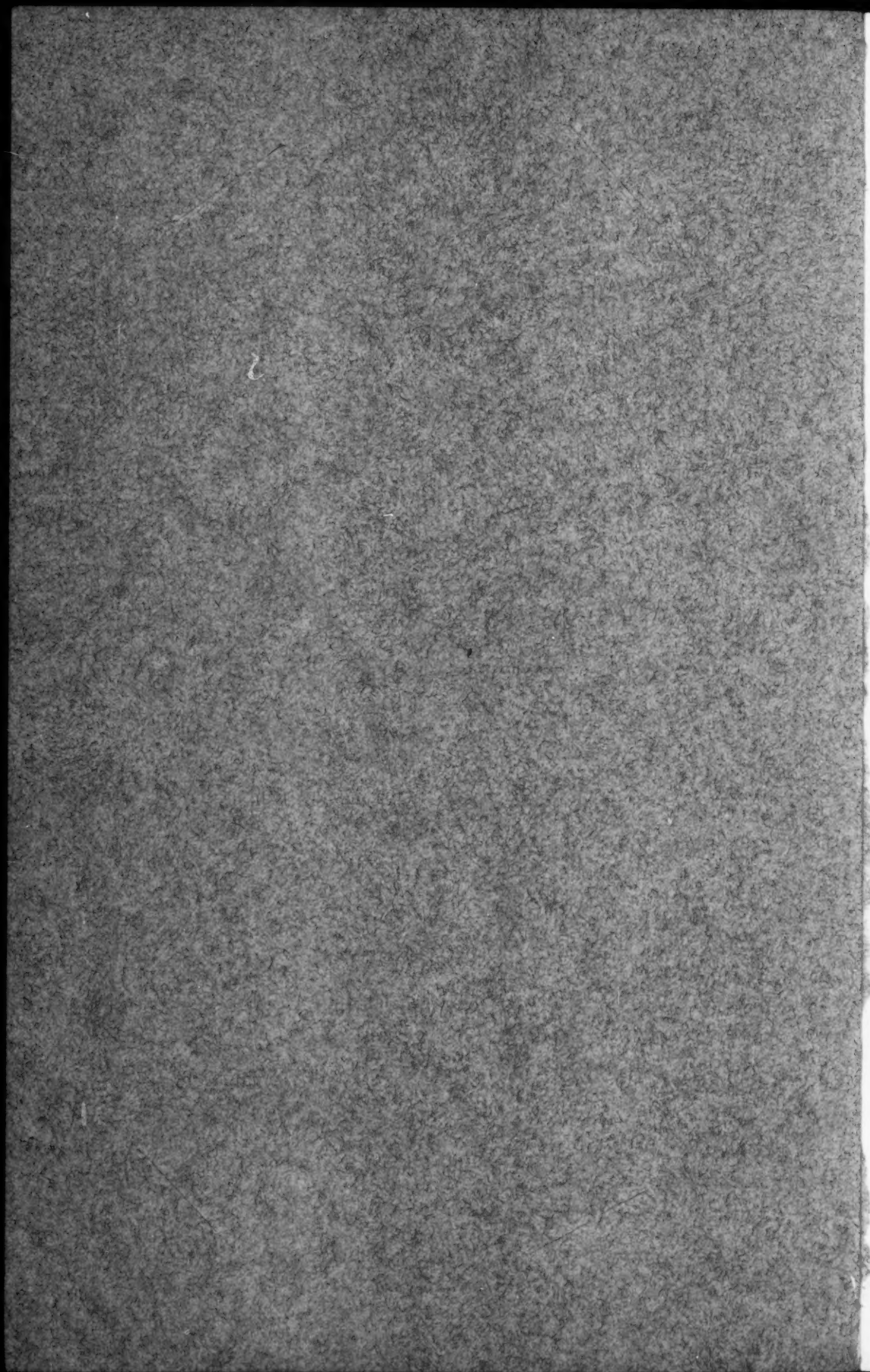


FIG. 5.



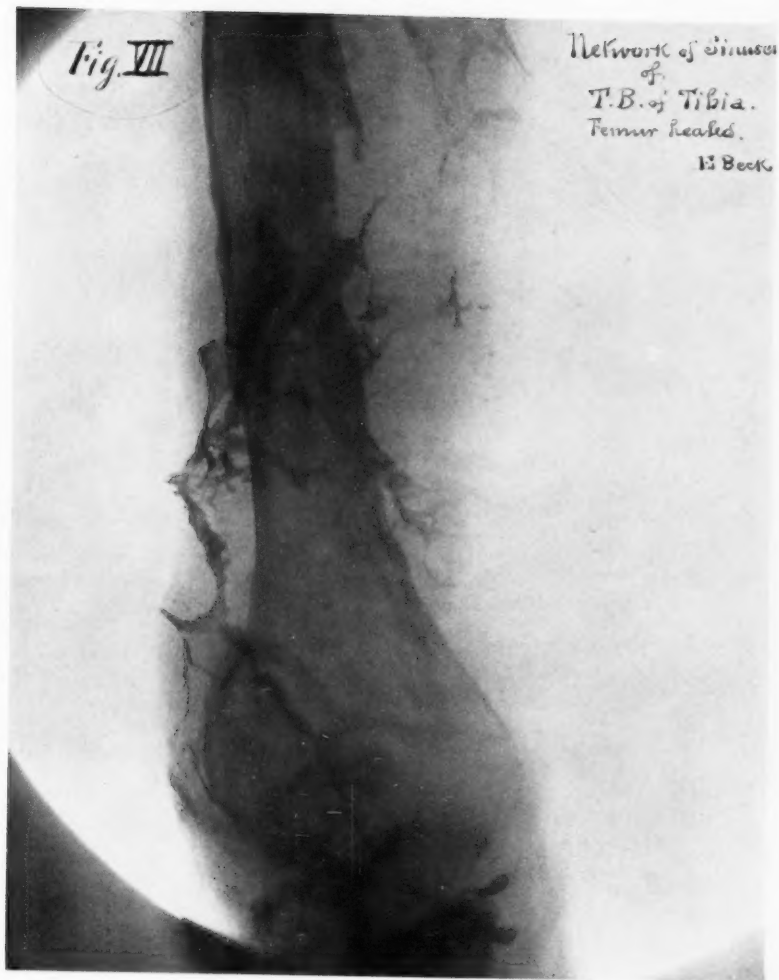
Supposed rectal fistula repeatedly operated, causing incontinence and invalidism disclosed to be tuberculosis of eleventh and twelfth dorsal vertebrae.

FIG. 6.

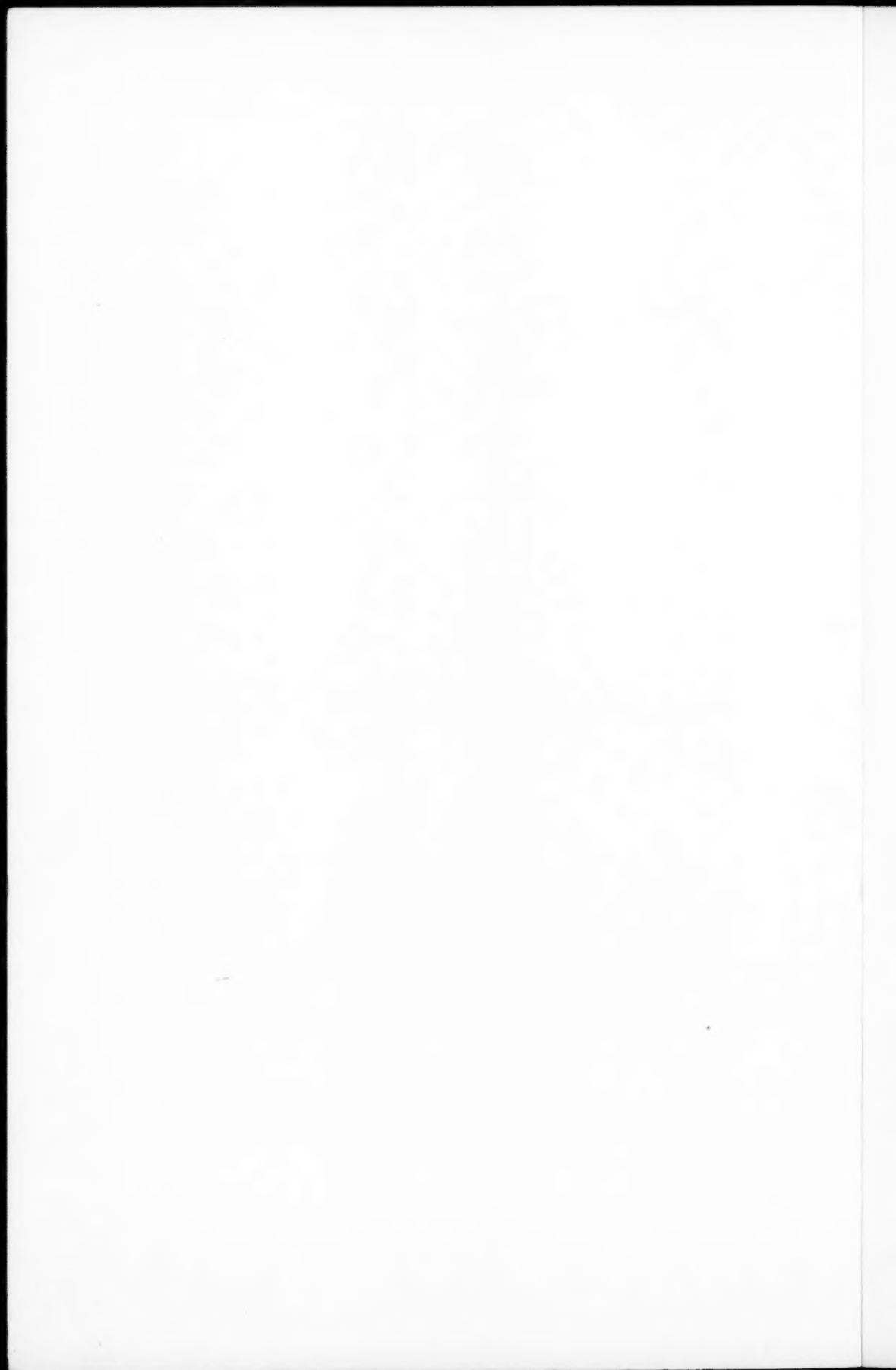


Hip-joint disease, sixteen years duration, operated fifteen times without success. Injection of paste in 1907, closure in thirty days. No recurrence.

FIG. 7.



Complexity of sinuses of knee-joint proving the uselessness of probing.



even the smallest percentage of cures in this series as an actual gain. The accumulation of reports in the past five years from all sources indicates that more than 50 per cent. of these apparently hopeless cases were finally cured.

The uses of bismuth paste are threefold, namely:

1. For *diagnostic* purposes, by which I mean for making a correct anatomical diagnosis and tracing the sinus tract.
2. For *therapeutic* purposes in chronic suppurations.
3. For *prophylactic* purposes, which means for the prevention of sinuses.

FOR DIAGNOSTIC PURPOSES.

Its diagnostic value has been acknowledged by all who have employed it. There are recorded in literature from all parts of the world many hundreds of cases in which this method cleared up puzzling conditions. For illustration, I cite here a case which is now under my care.

P. S., forty-one years old, rheumatism at twenty-nine, malarial fever at thirty. Ten years ago he developed a pronounced pulmonary tuberculosis and went to Arizona and gradually recovered from the tuberculosis of his lungs. January, 1910, he noticed a small abscess on the left of his sternum. It was incised and the bone scraped several times. In August, 1911, I removed a part of the sternum to eliminate the focus of disease, but a foul discharge persisted. Another radical operation was performed six months later in Arizona, surgeon resected the greater part of his sternum and four inches of the sixth rib adjoining the sternum. Discharge increased and condition became much aggravated. He returned to Chicago, June, 1913. An injection of paste with the taking of a radiograph for anatomical diagnosis immediately disclosed the cause of failure. The seventh and eighth ribs, at the junction of the lower part of the sternum, were diseased. The radiogram plainly shows that the disease extended through the interior of the two ribs (Fig. 2). A resection of these two ribs proved conclusively the correctness of the diagnosis. The ribs appeared normal on their upper surface, but I was absolutely certain that the disease was located within the marrow of the ribs (Fig. 3), and therefore removed them.

After their removal the suppuration ceased and wounds closed by perfect healthy granulation.

Examples like this explain why surgical operations for these chronic suppurating sinuses often fail. A glance at the radiograph which represents injected sinuses teaches us how irrational it is to attempt a dissection of a net-work of sinuses which lead into an inaccessible region. In the light of these pictures the probing of a sinus will not appeal to those who wish to be consistent (see Fig. 4).

I have previously cited a large series of cases in which incorrect diagnosis of the sinus led to useless and even dangerous surgery, in which, aside from the therapeutic results, the paste cleared up the cause of failure. (I cite here only one typical example.)

Supposed rectal fistula, repeatedly operated causing incontinence, disclosed to be tuberculosis of the eleventh dorsal vertebra.

A. H., fifty years old, was first seen by me in June, 1913. He stated that four years before he had been treated for pararectal abscess, after he had suffered for nearly eight years with what was thought to be rheumatism of the back. The operation resulted in a fistula, fever and emaciation began from that time. He was confined to his bed **nearly all the time**. A second and third operation was performed with division of the sphincter resulting in complete incontinence of the rectum. Another operation was then performed above the crest of the ileum and two more sinuses remained. I saw him in June, 1913, at his home in Canada. He was unable to walk, having been confined to bed for the last fourteen months. July 12, 1913, he was brought to Chicago. The injection of paste as shown in Fig. 5 revealed the true diagnosis. The sinuses with several side tracks reached from the rectum directly into the eleventh and twelfth dorsal vertebræ, where the disease originated. The injection not only cleared up the diagnosis but had a marked therapeutic effect. The rectal sinus closed, and the man gained twenty pounds in six weeks, and was able to walk about five miles each day. He is now engaged in his usual work, but the two sinuses at the crest of the ilium still discharge small quantities of pus.

FOR THERAPEUTIC PURPOSES.

At the present time very few doubt the therapeutic effect of the bismuth paste in old chronic sinuses and empyema.

In our series of cases treated at the North Chicago Hospital we have used the paste in practically every variety of chronic suppurations. We have treated sinuses resulting from spondylitis, hip-joint disease, tuberculous knee, ankle, shoulder, wrist and ribs, osteomyelitis in all parts of the osseous system, including the clavicle and fibula; further, chronic suppurations of soft structures, such as sinuses after extirpation of kidney, broken-down tuberculous glands, rectal fistulæ, and sinuses following laparotomies. The accessory sinuses of the head and the fistulæ of the alveolar process have also been treated by this method by my brother, Dr. Joseph C. Beck. Only fistulæ of the gall-bladder, the pancreas and those communicating with the cranial cavity have, for obvious reasons, not been treated, with the exception of one case of biliary fistula referred to me by Dr. Robert Morris, of New York, in which I tried it and obtained a splendid result. I shall reserve a tabulated report of our series of 1100 for another publication, but our results obtained so far have greatly exceeded those reported in the past.

We have learned from experience, and by improving the technic as well as by avoiding complications, to cope with the most hopeless cases, so that the percentage of complete cures has increased every year.

Statistics.—In order to reach a just conclusion as to the therapeutic results, I do not propose to hold up my own results as a criterion, I prefer to quote the average from the cases recorded in medical literature.

It is impossible to estimate the extent of the use of this method, because the bulk of the cases come into the care of the office or the patient's home, and thus only a small fraction of treated cases are published. Some large hospitals in this country, and clinics abroad, as well as individual surgeons, have from time to time made reports of their experiences, the collective summary of which up to 1913 is as follows:

Name	Number of cases	Percentage of cures
Ochsner, Chicago.....	20, tubercular sinus	55
Ridlon and Blanchard.....	17, tubercular sinus	53
Beck, E. G., Chicago	192, collective report	64
Robitecksh, Minneapolis	9, tubercular sinus	55
Don, Edinburgh.....	6, tubercular sinus	17
Rosenbach, Berlin.....	4, tubercular sinus.....	50
Dollinger, Budapest	16, tubercular sinus	12½
Beck, J. C., Chicago.....	319, accessory sinuses.....	22
Pennington, Chicago	17, rectal fistulæ	76
Baer, Baltimore.....	12, tubercular sinus	33½
Stern, Cleveland	4, tubercular sinus	100
Steinman, Muenchen.....	5, tubercular sinus	20
Bogardus, U. S. A.	1, tubercular sinus	100
Vidakovich, Russia	2, empyema	100
Nemanoff, St. Petersburg.....	6, empyema	100
Ochsner, A. J., Chicago	14, empyema	85
Beck, E. G., Chicago	11, empyema	82
Ely, New York.....	14, tubercular sinus.....	43
Hines, Cincinnati.....	9, tubercular sinus.....	89
Cuthbertson, Chicago.....	1, intestinal fistula	100
Sandoe, Sag., Budapest	2, otologii	100
Heitz, Boyer, Morens, Paris	11, renal sinuses	73
Zollings, Zurich.....	25, tubercular sinus	54
Schober, Philadelphia.....	5, tubercular sinus	80
Gessner, New Orleans.....	4, tubercular sinus	50
Schmid, Vienna	15, tubercular sinus	30
Rivero, Porto Rico.....	8, tubercular sinus	75
Goror, E., Paris.....	2, tubercular empyema	66
Reichelfelder, Washington.....	4, tubercular empyema	75
Brandes, Kiel.....	29, all varieties of sinuses	76
Beck, R., Chicago	58, alveolar sinuses.....	54
Beck, R., Chicago	9, empyema antrum.....	66
Collective reports from 19 dental surgeons in U. S. A.....	39, alveolar sinuses.....	74
Collective reports from 19 dental surgeons in U. S. A.....	4, empyema antrum.....	100

It must be taken into account that this represents a class of cases in which other treatment had previously been applied and had failed, some cases had even passed through a series of as many as twenty unsuccessful surgical operations, often the disease having lasted many years, yes, as many as forty years, as occurred in two of my cases.

An example or two may serve as an illustration of the therapeutic possibilities.

Hip-joint disease of sixteen years' duration. Fifteen operations. Injection of paste. Closure in thirty days.

Miss M. G., aged twenty-one, developed a painful condition

of her right knee and hip at the age of six. For one year she was treated symptomatically, and then a diagnosis of hip-joint disease was made by aspiration of pus from the hip. Incision and drainage (at the time considered the proper procedure) made. Condition was thus aggravated, and after six months of extreme suffering, often requiring chloroformization during dressings, a radical operation was performed, consisting in the resection of the head of the femur. This radical procedure, however, resulted in the formation of many sinuses and the persistence of fever. During the following ten years she submitted to thirteen more or less radical operations, at intervals of from six months to three years, all of which, however, were of no avail. The discharge and pain persisted. The last operation was performed by Professor Senn, in June, 1907. It was the most radical procedure thus far undertaken. Both trochanters were removed, and the acetabulum was thoroughly curetted. The five sinuses, however, kept on discharging pus. In December, 1907, the first injection of bismuth paste was made, and repeated every two or three days, and on January 15, 1908, the sinuses were closed and have remained thus for 6 years. The radiograph (Fig. 6) shows the extreme destruction of the joint, the end of the femur, including both trochanters, having been removed. The rim of the acetabulum is filled with bismuth paste, showing distinctly a collateral sinus.

Causes of Failure.—To explain the varying results among different authors, many factors must be considered. During my visits to various hospitals and clinics in America and Europe, I have had some opportunity to observe why some men have failed to obtain the best results and I shall enumerate the causes which I believe are responsible for the failures.

We must bear in mind that a sinus or fistula is nothing more than a shrivelled abscess or abscesses. No sinus originates by burrowing its channel from one end to another. The focus of the disease is often at a considerable distance from the opening or openings of the sinus. It is, therefore, inconsistent to try to eradicate the suppuration by only dissecting the sinus tracts. With the radiographic reproductions of the

labyrinths of sinuses before us, an attempt to dissect the same borders on the ridiculous (Fig. 7).

To strike at the root of the trouble is the only rational method to pursue. Find the focus from which the sinus originated, eradicate this focus and in practically all instances it will heal spontaneously.

It is, therefore, essential that when a fistula or sinus is injected with bismuth paste, the paste must reach the focus of the disease. If through faulty technic this is not done, one cannot expect results. It is likewise essential that all branches and crevices of the sinuses of the tract should be completely filled at one time. If one misses a part of the tract, the sinuses will continue to suppurate.

My brother Carl and I have treated a large number of cases in which the bismuth paste had been applied by others without success. This gave us the opportunity to study the causes of failure in a variety of most instructive cases. In some of these, the cause of failure could not be explained. The sinuses often healed after our first injection, whereas the same patient had received many injections previously with no result. We could not tell whether faulty technic, unsuitable instruments or improper material had been employed.

In other cases the causes of failure were easily discovered. Aside from the most common cause, namely, the sequestrum, we found foreign bodies, such as in one case a metal probe in the humerus; in another a rubber tubing within the medullary canal of the humerus, accidentally left in years before; then two rubber tubes within an old drained empyema cavity, etc. These were causes which had prevented the paste from obliterating the suppuration. As soon as these foreign bodies were removed, the cure was almost spontaneous.

These are, however, less common occurrences. They do not account for any considerable percentage of failures. From observation, I conclude that the most common cause is the faulty technic and insufficient knowledge of the rules which have been laid down for the treatment. I have been asked innumerable times: "How often do you inject?" The

proper answer to this question would be: As often as you have failed to reach the focus of the disease.

The first injection should produce the desired result. If it does not, then we must assume that the paste has not found its way into all portions of the diseased tract, and we must try it again. It is a safe rule to wait at least one week. If the discharge changes its character from purulent into a serous, and the microscopic examination of a slide and culture shows that the secretion is sterile, we should *not reinject*, since the sinus will heal out within a very short period. If, however, the discharge continues to be purulent, and we continue to find microorganisms in it, then we should reinject at least two times a week. If there is no change within a reasonable length of time, then we should search for other causes of failure.

Only six per cent. of all the cases in our series have finally been given up as hopeless. For obvious reasons we are not to be envied for the class of cases which fall into our hands for this treatment. They constitute the worst scum of surgical refuse, cases in which every sect of medicine, surgery and quackery had had a chance, and after all had failed to cure the sinuses then someone tried the bismuth paste. If this cured the case, all was well, but if it did not, then the case came to us. Nevertheless, out of this apparently hopeless material I am now able to exhibit many most satisfactory results. It is a mistake in giving a method only a superficial trial, to lose patience and subject the patient prematurely to another useless operation. Experience teaches us that operative treatment of this class of cases is highly unsatisfactory; if it were satisfactory, we would not have armies of invalids, with suppurating sinuses, going around from clinic to clinic some for as many as twenty years.

Surgeons all over the world are on the alert and would not permit such unfortunate people to linger in such condition if they knew of a surgical method for its eradication.

FOR PROPHYLACTIC PURPOSES.

By this is meant the prevention of sinuses. We know that the sinuses are the sequelæ of pre-existing abscesses, and therefore, we must begin by treating the abscess in order to prevent the sinus. This procedure consists in the opening of cold abscesses and injecting them at once with a *ten per cent.* bismuth paste, without suturing the opening or introducing a drain. The quantity used depends upon the size of the abscess, but should not exceed 100 grammes, because in these fresh abscess walls, absorption of bismuth is apt to take place more rapidly, and cause bismuth poisoning. The injection acts as a modifying substance, similar to that of iodoform emulsion, and prevents secondary infection. In a series of over 100 cases, in which I have employed it, only one developed a severe secondary infection, and only four resulted in sinuses. (No deaths.) Follins' figures show that 50 years ago, 56 per cent. to 60 per cent. of all psoas abscesses operated upon died from secondary infection. This method is described in detail in the *Revue de Chirurgie*, T. xlii, December 10, 1910.

We have treated with this method 110 cases, and have made the following observation:

(a) That in practically all cases we could prevent a secondary infection.

(b) That the creamy pus upon opening the cold abscess was changed into a straw-colored clear fluid within three or four days after injection.

(c) That 90 per cent. of all cases closed within three weeks after incision and injection.

The preference to the bismuth over other modifying substances was given for the following reasons:

1. The paste is injected through a small incision instead of using a trocar, and thus the possibility of missing the abscess is eliminated.

2. By discarding the aspirating needle the danger of

injuring underlying vital organs or entering blood-vessels is avoided.

3. Through an incision it is possible to evacuate the larger clumps of the tuberculous débris, which could not pass through the aspirating needle.

4. The thick paste within the cavity will allow the escape of secretions along the walls of the abscess, but will not permit the entrance of infectious material; thus secondary infection is prevented.

5. Injections of other modifying fluids must, as a rule, be repeated, while with the paste the first injection usually attains the desired result.

6. The injection of bismuth paste is not painful or irritating. It is injected in a warm, semiliquid state, and remains long enough in contact with the diseased tissues to produce its therapeutic effect. The vehicle (vaseline) does not macerate the walls of the abscess. Toxic effects from bismuth subnitrate can easily be prevented.

DANGERS AND COMPLICATIONS.

The only danger which has been advanced is the possibility of bismuth poisoning. My brothers and myself are fortunate in not having had a single fatal case in our series of cases. We met with the symptoms in one of the first cases of empyema treated, and were able to check the progress and save the patient. This case was reported by me in the *Journal of the American Medical Association*, January 8, 1909, and is the first case on record. I then warned the profession against the indiscriminate use of the paste. It is fortunate that most of these accidents were at once reported in the literature; this has put on guard those who thought that bismuth was an entirely harmless substance. It must have had a very salutary effect, because nearly all the cases of poisoning occurred in the first two years, 1908 and 1909, and the past year only one case is reported, although the bismuth paste is now employed among the majority of American surgeons, and to a large extent abroad.

It is gratifying to know that the poisoning can be prevented, and if it accidentally occurs and is discovered, it can be checked before it causes irreparable damage.

PREVENTION OF BISMUTH POISONING.

The prevention consists of not allowing large quantities of the paste to remain in the body for absorption. Should the symptoms appear, the paste must be removed by washing out the cavity with warm olive oil. The sterile oil is injected and retained for twelve to twenty-four hours, in order to produce an emulsion, which should be withdrawn by means of suction. After its removal all symptoms will promptly disappear. Scraping out the paste with a scoop is a dangerous procedure, because it opens fresh channels for absorption.

To insure success in employing bismuth paste the essential points are summarized as follows:

1. One should make a correct diagnosis by all methods at our disposal and corroborate same with stereoscopic radiographs before an injection is made.
2. Before attempting to employ this method, one should acquaint himself thoroughly with the technic.
3. The proper instruments should be employed in order to carry out the technic correctly.
4. The patient should be kept under constant observation to prevent bismuth intoxication.
5. Examine the secretions from the sinus before the first injection, by slide and culture, and often by the inoculation of guinea pigs; then three days later test the sterilizing effect of the injection.
6. As long as the sinus contains microorganisms it should be reinjected, but if it is found sterile, it should not be re-injected.
7. It is good practice to wait at least one week after the first injection before repeating it.
8. A stereoscopic radiograph of the parts affected should always precede the first injection, in order to detect the

presence of sequestra or foreign bodies. The shadow of the paste might make their presence obscure.

9. Following the injection, a second set of stereoradiographs should be taken in order to make a correct anatomical diagnosis.

10. In case a foreign body or sequestrum is present, the injection is useless, operation the only means.

11. Acute suppurative processes should not be treated with bismuth paste, only chronic suppurations, both tubercular and non-tubercular.

12. Bismuth poisoning may be easily prevented by using only small quantities, or when large quantities are required they should not be retained longer than ten days, and patient should be carefully watched.

13. Fecal fistulæ and other post-operative sinuses are very favorably affected by bismuth paste treatment.

14. A ten per cent. bismuth-vaseline may be used in cold abscess. In practically all instances the secondary infection can be prevented, providing the technic is carefully observed.

THE NATURE OF SHOCK.

ITS RELATION TO ACAPNIA AND TO CHANGES IN THE CIRCULATION OF THE
BLOOD AND TO EXHAUSTION OF THE NERVE CENTRES.*

(From the Laboratories of Physiology and Experimental Surgery of the
University and Bellevue Hospital Medical College.)

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It is the object of this paper to present the results of a series of experiments which furnish information regarding the relative etiologic importance of acapnia, of reflex changes in the circulation of the blood and of exhaustion of the nerve centres in shock produced by three methods. Crile has concluded as a result of a long series of experiments that the primary change wrought by all causes of shock is a fatigue of the vasomotor centre. As a consequence of this fatigue there is a continuous lowering of the blood-pressure until the cerebral centres, particularly the medullary centres, no longer receive sufficient blood to enable them to functionate normally, and from this cause, in fatal cases, life becomes extinct.

This theory has received the recognition to which it is entitled by the high character of all the work of its chief advocate. The protocols of his experiment demonstrate the close relation between low blood-pressure and shock. At present the theory is made the basis of a method of anæsthesia which is believed to diminish shock in operations, and is rendered conspicuous by having been christened with a new name.

No one can fail to admit the important association of a diminution of blood-pressure with the onset and development

* Read in the Section on Pathology and Physiology of the American Medical Association, at the Sixty-fourth Annual Session, held at Minneapolis, June, 1913.

of shock. Every writer has felt obliged to admit its bearing on the problem of shock. Nevertheless, a rather large number of writers since the publication of Crile's work have been unable to believe that any of the series of events, including the low blood-pressure itself, which may result in shock is, at the start, a process of fatigue of the nerve centres. Meltzer has discussed the whole question at length in a review of all the more recent theories of the nature of shock. He presents many observations of others and adds the results of his own experiments to show that the primary changes in the human body leading to the development of shock is not fatigue of the nerve centres but an inhibition of their activities. Differing from Crile, he quite justly questions the legitimacy of distinguishing etiologically between shock and collapse.

During the past winter we have performed experiments in connection with the production of shock by three methods. These experiments in agreement with the views of others, particularly of Howell, Porter and Meltzer, demonstrate, first, that a low blood-pressure is an important symptom of shock, but that an animal may pass into shock with a blood-pressure which is still far above a point below which the nervous system fails to functionate normally; and, second, that changes in the frequency of the heart and its output per beat always accompany shock, even in the earliest stages; but that it is very unlikely that changes either in the blood-pressure or in the force and output per beat of the heart are inaugurated by fatigue of the nerve centres. Our experiments indicate that shock in its incipency in some cases is of reflex, and in other cases of local peripheral origin.

From the practical as well as the scientific point of view the causes inaugurating shock are the most important. It matters little that a low blood-pressure may cause the death of an animal already in fatal shock. We wish to know what are the causes leading to shock before the blood-pressure begins to fall, and how to prevent these causes from becoming active.

Yandell Henderson has sought an explanation different from that of all other writers. He concludes from a large

amount of experimental work that acapnia can be one of these causes. He believes it to be the essential cause in shock produced by artificial hyperrespiration and by prolonged exposure of the intestines, and may be a cause of that form of shock produced by severe stimulation of afferent sensory nerves.

In all experiments performed by us, dogs were used. Each animal received 0.005 mg. of morphin for each kilogram of body-weight. Sufficient ether was given during each experiment to produce full anæsthesia.

SHOCK AND ARTIFICIAL HYPERRESPIRATION.

In the first series of experiments that form of shock was considered which was produced by artificial hyperrespiration.

A suitable time after the injection of the morphin the animal was etherized and a rubber tube introduced through the larynx into the trachea. By means of two rotary blowers and an intervening slide-valve which regularly threw first the exhaust of one blower and then the intake of the other blower into connection with the intratracheal tube, air was alternately forced into and sucked out of the lungs. The slide-valve was operated by another electric motor than either of those turning the blowers. Its speed was regular and could be controlled by a rheostat, and the frequency of the interruption of the valve which it operated could be varied as occasion demanded. By means of by-passes in series with the exhaust of the blower used to inflate the lungs, and the intake of the blower used to deflate the lungs, the amount of air used for the artificial inspiration and expiration could be varied at will. In these experiments the intratracheal catheter did not tightly fit the trachea. Provision, therefore, was made for the escape of any excess of air forced into the lungs during the inspiratory phase around the intratracheal tube, and the same factor of safety controlled the expiratory phase. We found, without this provision for a certain latitude in the amount of air used to inflate and deflate the lungs, that sudden death from excessive variations of pressure within the chest could occur. As will be developed, this fact is in accord with our own belief as to the cause of shock produced by excessive artificial respiration.

In four of these experiments which we have performed, the artificial respiration varied from 60 to 70 times a minute, and the lungs were as completely inflated and deflated as is possible with a closed chest. In order to accomplish such a filling and emptying of the lungs at a rate of from 60 to 70 times a minute, the air must be forced into and sucked out of the trachea under considerable pressure. Two of the experiments were continued for three hours and two for two hours. In these experiments the blood-pressure fell 40 per cent. within a few minutes after starting

the artificial respiration and then decreased more slowly to between 40 and 50 mm. of mercury. After the cessation of the experiments, the blood-pressure rose from 60 to 90 per cent. within a few seconds.

The carbon dioxide content of the arterial blood at the end of the experiments was from 38 to 44 per cent. of its original amount. The amounts of carbon dioxide and oxygen were measured in all experiments reported in this paper by the Barcroft-Haldane method. At the end of the experiment the animals were in deep shock. One died the next morning, one in two days and the other two lived three days. None of them died of the immediate effects of the experiment, but from secondary effects. They had recovered from the shock and their lungs at necropsy showed interstitial emphysema. In all these experiments it was found that the amplitude of the pulse and the blood-pressure was proportional to the pressure at which the lungs were inflated and, therefore, to the intrathoracic air-pressure. The amount of shock which was produced was proportional to the length of time that certain pressures, which we may term critical intrathoracic pressures, were maintained. The carbon dioxide content of the arterial blood could be easily reduced to from 40 to 50 per cent. of its original amount within half an hour; but in four other experiments when artificial respiration was maintained for only this short period, we found that no shock resulted. It has been assumed by Henderson that a long-continued acapnia, lasting two to three hours, results in a depletion of the tissues' store of carbon dioxide by osmosis, and accompanying this osmosis of carbon dioxide from the tissues into the blood, water passes from the blood into the tissues. As a consequence, a diminution of the total volume of the blood ensues. The associated general muscular relaxation dependent on the changed chemical composition of the muscles contributes to the diminution of the general blood-pressure by no longer affording the proper support to the veins. Thus the venopressor mechanism is also disturbed and, with it, the proper balance of the distribution of the blood in the body.

In our experiments the force of the artificial respiration necessary to produce acapnia was so excessive and the degree of shock and the change of blood-pressure so closely proportional to the intratracheal pressure that the air-pressures, at which the artificial respiration was given, seemed to us to be the most important factor in the production of shock by this means. We therefore performed three experiments in which the same conditions of artificial respiration were maintained, but with the provision against the loss of carbon dioxide. In all three precisely the same conditions of artificial respiration were maintained as in the first set of experiments, but, by inserting a rebreathing bag in which the expired air was collected and from which that blower was supplied which furnished the

air to the dog's lungs, and supplying to the fresh air required to be added during the experiment a proper proportion of carbon dioxide from a tank, the amount of oxygen in the blood was unchanged at the end of the experiment and the amount of carbon dioxide raised only slightly above the normal. All of these animals presented the same degree of shock at the end of their period of artificial respiration (two hours) as the animals of the first series. In these experiments, also, the shock was directly proportional to the air-pressures used during the artificial respiration.

Clearly, then, the shock produced by artificial hyperrespiration was not due to a diminution of carbon dioxide but to some other factor which is dependent on increased intrathoracic pressure. Of the effects produced by increased intrathoracic pressure, the one first suggesting itself as the most important and, as far as we can conceive, the only one bearing on the problem of the cause of this form of shock, is the interference of the venous return to the heart. By venous return to the heart, not only the return from the systemic circulation is referred to, but also the return from the pulmonary veins. The latter is affected in two ways, first, by direct pressure around the pulmonary artery, and second, on capillaries and veins within the lungs themselves. The pressure on the pericardium required to alter the general blood-pressure is much above that which affects the circulation when applied to the great veins at the base of the heart. This factor, therefore, can be neglected in these experiments. The most direct manner of measuring the effects of increased intrathoracic pressure on the circulation is to measure the output of the heart; and a third series of experiments, four in number, were devoted to this investigation.

The thorax was opened laterally, and a T-tube connected with a water manometer was tied in a small bronchus. The heart was then enclosed in a Henderson's cardiometer in circuit with a recording tambour. The blood-pressure was recorded from the carotid artery. The thorax was then closed and the animal subjected to intratracheal insufflation from an apparatus provided with an exhaust-valve which reduced the pressure to approximately zero from four to twelve times a minute, or

could be made to furnish continuous insufflation. In one experiment taken as an example, with an increase of intrabronchial pressure from 8 to 30 mm. Hg, the blood-pressure sank from 122 to 55 mm. Hg and the volumetric tracings of the cardiometer showed a diminution of cardiac output of 44 per cent. In another experiment the blood-pressure rose 15 mm. Hg each time the interrupting valve reduced the intrabronchial pressure from 6 mm. to 0. These variations of blood-pressure were completed within a few seconds after the change in intrabronchial pressure and could be duplicated at will. A rise of intrabronchial pressure above 8 or 10 mm. Hg always caused a fall of blood-pressure proportional to the rise of intrabronchial pressure.

It is evident, therefore, that excessive intrabronchial pressure, such as always accompanies violent artificial respiration even at from sixty to seventy times a minute, is quite sufficient in itself to account for a continued diminished cardiac output and low blood-pressure.

VENTILATION OF THE ABDOMINAL CAVITY.

We next studied the relation of acapnia to that form of shock produced first by exposure of the intestines to a current of warm moistened air passed over them beneath a celluloid cover, and second, by evisceration and handling the intestines.

A portion of the anterior musculature of the abdomen was excised, the omentum cut away and a celluloid window fitted in place between the layers of the muscles left at the side in such a manner as to completely cover the intestines. A current of warm and moistened air was then passed beneath the celluloid over the covered intestines. The air entered through a tube piercing the celluloid at one end of the abdomen and passed out through an opening at the other end. Aëration of the abdominal cavity under these conditions for a period of three hours produced no shock in one experiment and little reduction of the carbon dioxide content of the blood. Through the celluloid it could be seen that no drying of the peritoneal surface occurred. The intestine remained a good color, and peristalsis was almost absent at the end of this time. The blood-pressure was 163 mm. Hg. The celluloid membrane was then removed, the intestines spread out and the aëration continued for forty-five minutes longer. The blood-pressure was then 153 mm. and the carbon dioxide content of the arterial blood was 38.8 volume per cent. The intestines were then handled and in ten minutes the blood-pressure had fallen to 80 mm., and in twenty minutes to 56 mm. After even ten minutes longer there was 31.6 volume per cent. of carbon dioxide in the arterial blood.

As a check to this experiment another experiment was performed. The abdomen was opened by cutting away the anterior wall. The in-

testines were exposed by cutting away the omentum and warm, moistened air passed over them. A long tube was inserted into the trachea in order to preserve the normal amount of carbon dioxide in the blood. At the end of one and one-half hours the blood-pressure had not changed and the animal was in good condition. The intestines were then handled and in ten minutes the blood-pressure fell from 122 mm. Hg to 60 mm. Hg. The carbon dioxide content was 45.1 volume per cent. In twenty-five minutes the blood-pressure was 46 mm. Hg, the carbon dioxide still undiminished and the dog was in pronounced shock. The sciatic nerve was then stimulated and a rise of blood-pressure to 96 mm. Hg was obtained, showing a strong medullary reaction.

These experiments, investigating the relative effects of aerating the intestines and of handling them, justify the conclusion that the manipulation of the intestines and not a diminution of carbon dioxide is the important factor in the causation of shock accompanying exposure and handling of the intestines. We have been unable to find any record among the experiments of Henderson of the production of shock by aerating the abdominal cavity alone within reasonable lengths of time.

SHOCK AND MANIPULATIONS OF THE INTESTINES.

In attempting to investigate the mechanism of shock produced by prolonged handling of the intestines, we first sought to establish definite controls. After some preliminary experiments we demonstrated that by handling the intestines violently for one hour, with, it should be remembered, complete anæsthesia, a deep degree of shock could always be produced. In some of these animals the degree of handling of the intestines was sufficient to produce actual rhexis from the peritoneal surface. In our subsequent work we attempted to avoid such a severe degree of handling. We aimed to secure a very intense congestion without actual rhexis. We satisfied ourselves that this degree of handling, in two hours' time could be counted on to produce fatal shock.

Having established this fact we next attempted to discover how far it would be possible to resuscitate dogs from a condition of otherwise fatal shock produced in this manner by transfusion from another dog.

Deep degrees of shock were produced by handling the intestines in six dogs, as described before, for two hours. At the end of this time each animal was in a deep degree of shock. Their eyes were immovable in the orbits and drawn down and inward beneath the conjunctiva. They were absolutely irresponsive to sensory stimulation. Their muscles were relaxed, respiration was shallow, the surface of the body cold, and the pulse rapid and diminished in amplitude. In one of the dogs the transfusion was given immediately after the period during which the intestines were handled, in the others it was given at varying intervals up to one hour after the intestines were handled. Recovery from the shock followed transfusion in all of the dogs. In four of them immediately after the transfusion their eyes regained the normal position in the orbit. The recti muscles of the eyes recovered from their previous relaxation. The dogs voluntarily moved their legs and became responsive to external stimuli. Immediately after the transfusion three of them ran around the laboratory so that they were obliged to be tied up in order to keep them confined. Running around seemed to cause them no discomfort whatever. In two of the dogs which were not transfused until an hour after the experiment, and with which the blood-pressure had been allowed to reach a very low point during this hour, the recovery was less complete, though unmistakable. Following this improvement all of the dogs remained for a long time in about the same condition but permanent recovery was never obtained. They gradually manifested signs of increasing abdominal distress, becoming in consequence more quiet, and died some time during the following night. The temporary improvement after the transfusion described was only the well-recognized improvement regularly following transfusion in shock from any cause. Nevertheless we believe that these transfusion experiments on animals in shock from evisceration of the intestines afford information regarding the nature of shock when carefully studied themselves, and when taken in connection with the control experiments and other experiments about to be described.

The first significant fact to note, and one previously emphasized by Howell and Meltzer, is the comparatively high blood-pressure at the end of the period of intestinal manipulation. Only two of the animals had a blood-pressure approximating 50 mm. Hg. In all the other animals the reduction of the blood-pressure had been as follows: from 104 to 90; from 114 to 54; from 119 to 75; from 115 to 46, and from 105 to 80. The same failure of the blood-pressure during the period of the production of the shock to fall to a dangerously low point was noted in the control experiments; namely, from 116 to 84; from 118 to 67; from 110 to 94; from 102 to 90; from 145 to 88, and from 129 to 101. The one animal

which recovered ran around the laboratory in an apparently normal condition with a blood-pressure of 50 mm. Hg.

These facts demonstrate that at the end of the period during which the intestines were handled the nerve centres must have been supplied with sufficient blood to enable them to functionate properly in the absence of any other disturbing factor.

The second significant point was the very rapid recovery by the animal of his normal condition after transfusion. In other words, an animal in a deep degree of shock which our control proved would have certainly died in a few hours' time with a progressively falling blood-pressure, and in a number of instances with a blood-pressure which had already shown the first steps of this progressive fall, could immediately be resuscitated by transfusion. This rapid recovery precludes the idea that the other disturbing factor to which reference has just been made was an exhaustion of the nerve centres. We cannot conceive of an exhausted centre recovering so quickly. The fact that in our experiments the dogs spontaneously got up and played around and responded normally, as they did, to whistling, indicates that their cortical centres had not been exhausted by sensory impulses. There is no reason to assume that these impulses evoke a greater response in the medullary centres than in the cortical centres. Our deduction, therefore, that the medullary centres were not exhausted or even fatigued is justified. We draw no distinction except in degree between exhaustion and fatigue.

This conclusion is in accord with the results of Porter's experiments which furnish strong evidence that the medullary centres are not exhausted in shock. Porter obtained in numerous experiments a greater percentage rise of blood-pressure by stimulating the sciatic or vagus or splanchnic, or a greater percentage fall by stimulating a depressor nerve after the blood-pressure had been reduced in shock than before the shock had been produced. With a low blood-pressure the same strength of stimulus would probably be more effective both because the vessels may be dilated and because their walls

meet less resistance during contraction. Nevertheless, the absolute rise or fall in Porter's experiments was very great and the experiments furnish strong evidence of the absence of fatigue in the primary stages of shock.

In one experiment we have confirmed the results of Porter's work. A dog was thrown into deep shock by one and one-half hours of violent artificial respiration. On afferent stimulation of the vagus, or sciatic, or stimulation of the splanchnic, a percentage rise of blood-pressure of almost 100 could be obtained. The absolute rise was practically the same as at the beginning of the experiment before the shock had been produced, namely, 30 mm. Hg.

Those who have explained shock as primarily an exhaustion of the nerve centres assume that the blood-pressure in an unconscious animal falls because the medullary centres respond to afferent sensory stimuli and thus dissipate their energy. Numerous experiments have been reported by others in which animals have been thrown into deep shock by prolonged crushing, tearing, and electrical stimulation of sensory nerves. The results of these experiments have been interpreted as demonstrating the power of prolonged and strong afferent stimulation to exhaust the nerve centres. They have been used to explain the shock following serious injuries or operations and of the various methods of producing experimental shock. If, however, these results are used to interpret other forms of shock, they should parallel, particularly as regards time, the actual conditions of the accidents, operations or experiments which they are used to explain.

We have performed experiments of this kind. The animals have received the usual dose of morphin which has been used in all the work presented in this paper. They were then etherized. The sciatic and brachial nerves were dissected out and a strong faradic current applied for two hours to the nerves. Much tearing and crushing of the nerves was incidental to the experiments. During the period of stimulation the medullary centres were certainly active and presumably dissipating energy. This was proved by the hyperpnœa and rise of blood-pressure maintained during the experiment. As soon as the stimulation was discontinued there was a definite fall of blood-pressure, never, however, to a degree which either indicated shock, or could be of any significance in its pro-

duction. The blood-pressure averaged, for instance, at the start of the experiment, during the period of stimulation and after the latter was discontinued respectively 150, 120 and 110 in the first animal; 90, 120 and 100 in the second; 130, 176 and 140 in the third, and 96, 116 and 74 in the fourth.

These dogs required considerable ether, which regularly lowered the pressure each time it was applied. At the end of the experiment all four dogs recovered promptly. Immediately after the experiment the frequency and amplitude of the pulse was good. It compared favorably with that at the beginning. In one hour's time one of the dogs responded normally to his environment; the other three in four hours' time.

There was certainly little difference in this manner of recovery from that which would be presented by another animal which had received an equal amount of morphin and ether.

These statements are emphasized by the differences presented by animals in which the same prolonged severe stimulation of the sciatic and brachial nerves was conducted after the animal had lost the power of controlling his blood-pressure by a preliminary division of the great splanchnic nerves. Three of these experiments were performed. In one animal at the end of fifty minutes' stimulation the blood-pressure had fallen to 14 mm. Hg, death following a short time later. The second withstood a continuous stimulation for two hours; at the end of this time the blood-pressure was 77 and the animal was in deep shock; in three hours' time he was in still deeper shock and he was killed in five hours' time. The third animal recovered from the immediate effects of the experiment.

The relation of diminished blood-pressure to the production of shock in association with the stimulation of sensory nerves was intensified by bleeding the dogs after the splanchnics had been divided. One of these experiments was performed after division of both splanchnics, 200 c.c. of blood were withdrawn, reducing the primary blood-pressure from 152 to 70. The sciatic and brachial nerves were then stimulated as in the preceding experiment. The animal died in deep shock before the conclusion of the experiment.

In four other experiments dogs were bled until the blood-pressure fell to a degree comparing favorably with the fall produced by dividing the splanchnics and the sciatic and brachial nerves were then stimulated for two hours. All four

of the animals developed deep shock; one of them recovered with the aid of an infusion and was alive the next day; another recovered spontaneously, though he did not stir when disturbed; another died during the experiment from excessive anæsthetization, and the fourth succumbed from the experiment. On the other hand, animals subjected to similarly caused reduction of blood-pressure and equal periods of anæsthetization by ether, but not to the prolonged sensory stimulation, suffered from a degree of shock which we were unable to distinguish from that of the stimulated animals which were similarly bled. It must be remembered in this connection that the latter required more ether. We have performed three such control experiments and are satisfied as to the truth of this statement.

As soon, however, as the animal's blood-pressure was reduced and the animal was deprived of his power of compensating for lowered blood-pressure by paralysis of the splanchnic area, serious shock developed but always in proportion to the diminution of blood-pressure and not greater than in animals in which the blood-pressure was reduced to a similar degree by hemorrhage alone.

In shock produced by prolonged handling of the intestines it seems that much less severe sensory impulses can be present than occur in stimulation of the sciatic and brachial plexus for the same length of time. Consequently, if sensory impulses in an unconscious animal were not effective in producing shock by causing exhaustion of the central nerve cells in the absence of vasomotor control, it is not likely that they are the important factors in the production of shock by prolonged handling of the intestines. Simple division of the splanchnic, as we ourselves have also experienced, does not in itself result in a lowering of the blood-pressure sufficient to produce shock. Within the time limit which we have adopted in these experiments, which is quite sufficient from the practical point of view of the operating surgeon, the exhaustion of the nerve centres by afferent stimulation of sensory nerves is a wholly negligible factor in the production of shock.

CAUSE OF SHOCK PRODUCED BY MANIPULATION OF THE
INTESTINES.

Returning again to the interpretation of the experiments in which shock was produced by prolonged handling of the intestines and in which attempts were made to resuscitate the dogs by transfusion, the quick recovery precludes the idea that the nerve centres had been exhausted. It does not, however, negate the possibility of the condition of the animals at the end of the period of intestinal handling being due to cerebral anæmia in combination with the anæsthesia which had been used. While the blood-pressure was still far above a level which would prostrate an animal wholly out of anæsthesia, there was little difference in the condition of these dogs and similarly anæsthetized dogs whose blood-pressures had been reduced to a similar degree by hemorrhage. Cerebral anæmia, however, is a far different condition from exhaustion of the nerve centres, a state demonstrated to be absent, as we have repeatedly emphasized, by the rapid recovery after transfusion.

But as has been stated, with the fairly high blood-pressures recorded in these experiments cerebral anæmia could have contributed little to the degree of shock exhibited by the animals. Many facts indicate that coöperating with the cerebral anæmia, inhibitory impulses are important causes of the animal's condition at the end of the period during which the intestines are handled. On the cessation of the handling there would be a return of a reflex response of the animal indicating semiconsciousness. An immediate relapse into an insensitive comatose condition could be produced by continuing the handling. The blood-pressure usually fell when the handling was stopped and rose again under the stimulus of handling, though this was not a constant effect. We know that afferent impulses of possibly an inhibitory nature are present. It is due to them that these experiments of evisceration and intestinal manipulation may be performed at times without the continuous use of a specific anæsthetic other than morphin and the preliminary anæsthesia, and yet without any evidence whatever of feeling on the part of the animal. There

is no physiological reason for distinguishing between shock and collapse. The latter condition is entirely due to inhibition. Howell and Meltzer have presented additional evidence that inhibitory impulses are important factors in this stage. They unquestionably are responsible in the beginning of the experiment for the onset of shock and the first fall of blood-pressure. If they are then operative they must continue to be during the whole time during which the intestines are handled.

Following the period during which the intestines were handled in those animals in which the shock was produced for control purposes and which were not therefore transfused, there occurred a progressive fall of blood-pressure within the next few hours until death occurred. The progressive fall was often initiated by a considerable drop at the start. This progressive fall was unquestionably due to bleeding into an absolutely paralyzed splanchnic system. During this period we have found that stimulation of the splanchnic nerves produced no rise in blood-pressure, or change in a plethysmographic tracing measuring the amount of blood in the splanchnic area. There were even no indications of blood-flow through the intestinal vessels.

The local peripheral character of this vascular paralysis has been clearly shown by two experiments. A coil of intestines was protected in a plethysmograph during a period of handling of the intestines for one hour and one and one-half hours. The splanchnic nerve was stimulated and the diminution of volume within the plethysmograph recorded before and after the period during which the intestines were handled.

The protected loop and kidney showed a marked change in volume both before and after the period of intestinal manipulation, while after this period a loop of the handled intestine showed no change.

There can be no question therefore about the extreme paralysis of the splanchnic area after two hours' handling of the intestines. There is an absolute paralysis of every tissue of the intestines, of the muscles, of the intestinal walls and of the arterioles. There is an absolute abolition of all reflexes. The great means by which vasomotor changes in the body are

possible, that which the vasomotor centre uses to produce its rise and fall of pressure and without which it is powerless, is hopelessly unavailable.

The amount of blood which this area will contain is well illustrated by a number of experiments which we performed, in which during the period of handling the intestines, the brain of the same animal was supplied with blood from the carotids of another dog, and in one case from the carotids of two other dogs. Before the intestines were handled, an anastomosis was made between the carotids and external jugular veins of the donor and the recipient, which was to be shocked. The purpose of these experiments was to discover whether or not any diminution of shock could be obtained by supplying the dogs being shocked, with blood from a presumably normally beating heart, thus eliminating the small fall in pressure occurring in the other transfused dog, during the experiment. In general the dogs gave the same result as the dogs transfused at the end of the experiment. The main purpose of the experiment was defeated, however, by the fact that the splanchnic area of the recipient during the period in which the intestines were handled drained off so much blood from the donor and in one case from two donors, that the blood-pressure of all donors in the three experiments fell to a serious degree, so that at the end of the experiments the donors no longer supplied the brain of the recipient with blood under good pressure, and were themselves in a serious condition from exsanguination.

Animals shocked in the manner described are deprived of all vasomotor control solely because of a local peripheral paralysis of the splanchnic area. It is as though the branches of their mesenteric arteries emptied into a large reservoir with perfectly flaccid walls, into which they bled to death. The aptness of the comparison of the splanchnic area to a flaccid rubber bag is made more apparent by pressure on the abdomen. The blood-pressure can be raised at will by this procedure. The explanation of the secondary shock developing in the transfused animals, the intestines of which are paralyzed from one end to the other, introduces very complex questions which

are not concerned in this paper. Suffice it to say that the animals remained in good condition with high blood-pressure in one case—the only dog watched till death—for twelve hours, and that they rather suddenly passed into a moribund condition.

CONCLUSIONS.

Our conclusions, which we hope to support by more numerous experiments, and by reporting them in greater detail in the future than has been possible in this paper, are as follows:

1. As severe a degree of shock may be produced by artificial hyperrespiration, and by handling of the intestines when provision is made for keeping the carbon dioxide content of the blood high, as when it is allowed to fall to 40 or 50 per cent. of the normal.

2. Shock produced by artificial hyperrespiration is due chiefly to a long-continued, mechanical interference with the return of the blood to the heart.

3. There is evidence that the early stages of shock produced by evisceration and handling of the intestines is due to inhibitory afferent impulses.

4. At the end of the period during which the intestines were handled none of the animals' nerve centres were exhausted.

5. By such handling of the intestines a complete splanchnic paralysis of local peripheral origin is produced, and it is this paralysis which causes the subsequent fatal fall of blood-pressure and not exhaustion of the nerve centres.

6. In the presence of a good blood-pressure and unimpaired vasomotor compensatory mechanism, prolonged afferent electrical stimulation for two hours will not produce shock or exhaustion of the nerve centres.

7. If trauma to the sensory nerves is a factor in production of shock in an unconscious animal, it is wholly subsidiary to other factors, and it is questionable whether it was apparent in our experiments even when these other factors had rendered the nerve centres more vulnerable by toxic influences, as ether, or by a fall in blood-pressure.

8. The all-important factor in the development of shock, in so far as the forms which we have studied may represent shock in general, is loss of vasomotor control. It is, at least, the impossibility of regaining this control after it has reached a certain degree which determines the failure to recover. The mechanism of this loss and its maintenance is important. The loss of control and its maintenance is never caused by acapnia or central nervous exhaustion, but, aside from afferent impulses more especially splanchnic sensory impulses which may have initiated the shock and contributed to it, the loss of control was always due to local peripheral causes which in our work were mechanical obstruction, loss of blood and trauma to the viscera.

The practical conclusions from these observations emphasize the necessity, in attempting to prevent shock, of providing against a fall of blood-pressure and local trauma, particularly within the abdomen, as the most important of all precautions. The truth of this statement at present is so generally acknowledged that it is almost trite to make it. Nevertheless the conclusions, indicated by the experiments in which unsuccessful attempts were made to produce shock by trauma to peripheral sensory nerves, will not be generally accepted. They directly contradict grounds on which the method of anaesthesia known as anoci-association is based. We appreciate that our experiments are few. Nevertheless, a study of their details demonstrates that their results were decisive and that severe trauma both electrical and mechanical of peripheral somatic nerves in an unconscious animal within reasonable time limits did not result in either a reflex fall in blood-pressure or exhaustion of the nerve centres. Its influence as a cause of shock at least in so far as the three forms of shock which we have studied may serve as examples of shock in general is so small that it may be practically neglected. In this connection it must be remembered that shock following burns is toxic in its nature.

However valuable the blocking of sensory nerves during operation may prove, the explanation is not to be found in the protection which it may insure against fatigue of the nerve

centres, certainly within the time limits of the usual operation. It is doubtless a wise precaution, on account of the more complicated manner in which reflexes may be modified in the human being than in animals, to block the larger trunks of the sensory somatic nerves when these must be divided. Clinical experience certainly teaches that it is most desirable to block the sensory splanchnic nerves when their trunks or more particularly the region of their plexuses must be subjected to trauma. Such blocking will often spare a patient reflexes which may seriously lower the blood-pressure. But the harmful effects, if it persists, is not due to fatigue of the nerve centres but entirely to reflexes and peripheral changes which may be either secondary to them or the result of other local peripheral causes or both. It is equally important to recognize that vasomotor control may be impaired or lost by peripheral injury alone. The central mechanism seems capable of outlasting the peripheral mechanism every time.

The necessity of guarding against loss of blood is self-evident. Of equal importance is the selection of an anæsthetic which, aside from any consideration of toxicity, does not reduce blood-pressure. Ether does not always fulfil this condition. Clinically and experimentally, unless administered with the greatest care, it strongly reduces the blood-pressure. We have numerous illustrations of this fact among our tracings and are disposed to attribute much of the shock of long operations under ether to this fact and to its toxic effect on nerve tissue and the glandular organs. Nitrous oxide does not possess this disadvantage and is also much less toxic. Crile has in no instance shown his keen appreciation of those factors which make surgery more successful than in his advocacy of nitrous oxide anæsthesia. If the general blocking of sensory nerves only increases the efficiency of nitrous oxide anæsthesia it is for this reason valuable. Its effect in eliminating harmful reflexes caused by trauma, particularly in the region of distribution of the splanchnic sensory nerves, has been explained.

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THE DIETETIC TREATMENT OF GANGRENE IN DIABETES MELLITUS.*

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THE purpose of this paper is to call attention to certain surgical conditions to which diabetic patients appear peculiarly prone and, since the usual operative procedures are notoriously unsatisfactory in their ultimate results, to draw attention to some factors which we believe are, in part at least, the cause of the failures, and to suggest a different mode of treatment and emphasize for these cases the importance of a proper diet. The conditions referred to are those changes which take place in the extremities, usually in the toes or feet, and which are classed as diabetic gangrene and more properly spoken of as gangrene in diabetics. The following histories will show that these are not always cases of gangrene, though often considered as such, but simply infections which run a course rendered peculiar by having occurred in diabetic subjects.

The surgeon frequently recommends immediate amputation in cases in which there is gangrene of a portion of the toes or foot and a pronounced interference with the circulation as evidenced by stasis, congestion, and hyperæmia of foot or leg. Too often the surgeon's attitude is that the treatment of diabetes is not within his province, and this serves as a cloak for his ignorance of what is a proper diet. If any one hopes to perform successful surgery on diabetic individuals it is absolutely necessary to treat their diabetes and this is best done by supplying a suitable diet. With the possible exception of the alkalis to combat acidosis the use of drugs is of no value.

The salient facts can be best set forth by the histories of the following cases:

* Read before the New York Surgical Society, November 12, 1913.

CASE I.—Admitted to the service of Dr. Charles H. Peck, at the Roosevelt Hospital, suffering from what was spoken of as “diabetic gangrene” of the foot. The patient, a man, had had diabetes mellitus for several years but had never had any proper treatment. Six weeks before admission a corn on the fourth toe of the right foot became infected. The infection developed into a cellulitis and gangrene of the fourth and fifth toes, and they had sloughed off prior to admission. On entering the hospital the patient was very ill. Temperature 104.4° ; pulse 120. Urine contained 7.1 per cent. of sugar and showed marked reaction for acetone and diacetic acid. The foot and leg were discolored and cold and the circulation barely perceptible. There was a marked degree of cellulitis and a collection of pus on the dorsal and plantar surfaces of the foot. The wounds were unhealthy and the sloughing tendons and strands of fascia were bathed in pus. The second toe was gangrenous and the line of demarcation was not clearly defined but merged into the surrounding cellulitis. At first amputation seemed to be unavoidable, but as the urine contained large amounts of sugar, and also gave a strong Gerhardt’s reaction, an extensive operation under a general anæsthetic was believed to be of hazardous outcome, because of the danger of coma. Accordingly the second toe was snipped off with scissors and under local anæsthesia suitable incisions were made to drain the pockets of pus on the dorsal or plantar surfaces of the foot, and sloughing tendons and pieces of fascia were pulled out and removed. The wounds were loosely packed with gauze tape, soaked in formalin, and a wet dressing of formalin applied to the extremity as high as the knee. The patient was then given a proper diet and was not sent to the ward and placed on “diabetic diet,” which in most institutions means a supply of nourishment selected by the nurse, which is said to be free from “sugar.” Oh that expression “diet free from sugar”! It is in most training schools almost as sacred as that other shibboleth, “peptonized milk by the cold process” and it is a brave surgeon indeed, who has the temerity to suggest that neither of them serves his purpose. This patient was placed on a suitable diet in the hope that the acidosis might sufficiently diminish to permit of the amputation. In the course of a few days not only did the ketonuria disappear, but the sugar excretion fell off markedly also and the foot and leg rapidly returned to

normal. The infective process diminished, the temperature fell to normal and the wounds under daily dressing healed slowly, the sloughs separating and granulations of a healthy character soon closing the wounds. At the end of a month the urine was free from sugar, acetone and diacetic acid, and the patient left the hospital with wounds healed, having escaped amputation and with his diabetes much improved. He continued on a strict diet; his diabetes was held in check until he died of cerebral apoplexy eighteen months later.

CASE II.—A Jewish woman, fifty-one years old, whom one of us (Dr. Foster) had seen several times in consultation. She loved food and especially sweets, detested dietetic restrictions and pretty well ignored her medical advisers. In June, 1912, an acute inflammation of one of the great toes appeared. The slight swelling, redness, and heat in this location, together with the pain, at first suggested gout. After a week, however, the color began to change to a livid hue which later became purplish. The increased heat disappeared and the toe grew colder than the others up to midtarsal joint, and insensitive to touch, although still somewhat painful. There was evidence of marked arteriosclerosis of the radial arteries. The condition was pronounced gangrene and operation advised. The diet had been largely of carbohydrates during the time when the gout theory of causation was entertained and the urine averaged about 200 grammes of sugar *per diem*, there was, however, no evidence of acidosis. It was determined to give diet a trial before proceeding to radical measures, and a competent nurse was put in charge. A strict dietary was carried out for three weeks before the urine was rendered sugar free. During this time the toes did not notably change either for better or worse; but after the urine had been sugar free about a week, the color of the toes began to fade, becoming white and finally normal flesh color. At the same time sensation and warmth slowly returned until a complete recovery was made. This patient remains well although the diet is only an awful memory and the urine contains sugar.

CASE III.—A man fifty-two years old, was admitted to the service of Dr. Joseph A. Blake at the Presbyterian Hospital, with the following history:

The patient remembers no illness prior to one year ago when he had a small blood blister on the fifth toe of his left foot. This

FIG. 1.



Case III. Note calcification of dorsalis pedis artery, marked periostitis of third and fourth metatarsals, and osteomyelitis of fourth metatarsal and phalanges of fourth toe.

broke and as it had not healed in three weeks a physician was consulted, who examined the urine and found it contained sugar. The terminal phalanx became gangrenous and six weeks later was removed under local anæsthesia. The patient was then placed on a general diet in which the sugar was restricted and for eight months had no further trouble. Fortnightly examinations of the urine showed 1.3 to 1.6 per cent. of sugar. At the end of eight months, that is two and a half months prior to admission, he noted another blood blister on the fourth toe of the same, the left, foot. Several weeks later a small piece of bone was discharged from the ulcer after which the wound healed. A superficial ulceration then appeared at the base of this toe on its plantar surface. This gradually increased in size during a month and a half until it extended on the plantar surface of the foot as far as the first toe. The patient stated that up to a month previously he had attended numerous banquets and did not restrict his diet. The sugar in the urine ran as high as 2 per cent. During the month prior to admission he had been on what he called a milk and egg diet almost exclusively with rarely an oatmeal day, and the sugar in the urine had averaged about 1 per cent. He had also been compelled to arise several times at night to pass his urine. He thought the amount secreted increased above his former daily average of two litres. His temperature during the month prior to admission averaged between 98° and 100°. His appetite was poor. Bowels were constipated and moved only by taking salts. He never had great thirst and did not lose weight, his average being about 288 pounds. He had no skin lesions and no respiratory symptoms. On admission there was a marked degree of cellulitis in the left foot and leg, with redness, swelling, pain and tenderness extending above the ankle. There was a sinus on the plantar surface which was lined by sloughing tissue and from which a small amount of pus with foul odor exuded. The sinus led to bare bone.

The X-ray (Fig. 1) showed a pronounced degree of necrosis and destruction of the fourth metatarsal bone and the first and second phalanges of the fourth toe. There was also evidence of a marked periosteal thickening of the shaft of the third and fourth metatarsal bones. There was evidence also of an osteoporosis in the bones of the other toes. A portion of the first and all the second and third phalanges of the fifth toe were missing. There

was calcification of the dorsalis pedis artery, plainly shown as a shadow between the first and second metatarsal bones.

The urine on admission was: total quantity, 1515 c.c.; sugar 3 per cent.; glucose 45.45 grammes; acetone moderate reaction; diacetic acid, faint trace; total nitrogen 12.41 grammes; ammonia nitrogen 0.89 grammes. The kidneys excreted 64 per cent. of phenolsulphonthalin in two hours. Although amputation of the foot seemed to be the only thing to do, it was determined to delay as it would not increase the danger to the patient and a trial of suitable diet was advised.

This patient had practically no ability to utilize any carbohydrate; and ingest of gms. 20 of starch was followed by the excretion of gms. 19.8 of sugar and it required three weeks before a sugar-free urine was secured. In this interval, however, the assimilation limit had been raised appreciably as was evidenced by the excretion of from 3 to 6 grammes of sugar after ingesting 20 grammes of starch. At first there was no evident improvement in the foot, but after two weeks a rather sudden turn for the better occurred and this was coincident with a marked return of the patient's strength and well being. Large pieces of plantar fascia came away as sloughs, the circulation in the foot improved slowly and the sinuses became lined with healthy granulations which subsequently healed, and at present he is walking about and reports that he feels "perfectly well." The urine now remains sugar free, although the present diet contains 160 grammes of starch.

The conditions illustrated by these cases raise an interesting pathological question. In the proper meaning of the term this morbid process is not a gangrene; that is not conceivable in the light of ultimate restoration to normal, and yet the condition cannot be differentiated from some cases of gangrene, as there is present a stasis of the circulation which is almost complete.

Many theories have been advanced as to the nature of the process and its underlying causes. There seem to be several factors, all or any combination of which may be present in a given case. There is, we believe, an infection with micro-organisms in every case and there is no specific organism but the common pathogenic forms. In addition to this there may

be marked arteriosclerosis, as in our third case, or a marked alcoholic diathesis, as in our first case. We believe that in certain cases there is present a process analogous to Raynaud's disease as suggested by the second case. Several considerations suggest that it is possibly the increased amount of sugar in the circulating blood which may have reduced the resisting power of the cells. If the last hypothesis be correct it would explain the amelioration of symptoms following successful dietary regulation, since this regulation lowers the percentage of blood sugar; which is its ultimate object.

It is not our contention that every case of "gangrene" in a diabetic patient is of the type here described. There may occur the "fulminating cases" in which high amputation is clearly indicated. But it is rational to give each case as thorough a course of dietary treatment as possible, especially as the results of surgical treatment are most unsatisfactory.

We wish to give the principles of the proper method of feeding these patients and will go somewhat minutely into the dietary.

What this dietetic regulation should be in a given case depends of course upon the type of the diabetes. The principle involved is the same in all. Excretion of glucose in the urine is purely an overflow of excessive blood sugar. Normally the glucose content of the blood is not above 0.1 per cent. In diabetes, however, it is often three or four times this, or even more. The kidneys do not hold back this excess, hence glucosuria. The object of dietetic treatment is to reduce the blood sugar to something near the normal, and the available measure of success in this attempt is the urine. Hence it follows, no diabetic is to be regarded as successfully treated so long as sugar is excreted.

The means at our command of combating this disorder are purely dietetic. It is necessary to restrict the carbohydrate ingest to an amount that is completely utilized by the patient. At the same time we have to remember that total withholding of carbohydrate for more than a few days at a time may also lead to injury to the patient. The problem then is to find the amount of starch to give.

First, with regard to those patients who present no evidence of acidosis, the urine gives no reaction with ferric chloride (Gerhardt's test). These cases may be divided for our convenience into classes: (1) Where the urine becomes sugar free quite promptly after restriction in carbohydrate; (2) where the urinary sugar falls to trifling amounts, 1 to 5 grammes per day, on restriction of starch, but fails to disappear completely on this diet. In both of these classes, without evidence of ketonuria, the diet may be reduced at once to very small amounts of carbohydrate by using meats, eggs, fats, and vegetables that contain little starch. The foods that are available for this diet make up Table I.

TABLE I.

Breakfast¹: Eggs, chops, broiled chicken, fish (fresh, salt or smoked), ham, bacon; tomatoes, onions, mushrooms (broiled or fried); coffee, 1 tablespoonful cream, saccharine to sweeten.

Lunch: Clear meat broths, meat of all kinds, game, poultry, fish; green vegetables, served hot with butter sauce, spinach, Brussels sprouts, string beans, asparagus, artichokes; salad of lettuce, endive, cucumber or tomatoes, with oil and vinegar, and any kind of cheese.

Dinner²: Clear broths, *e.g.*, consommé; meats same as lunch; artichoke root as substitute for potato, cabbage, asparagus, spinach, string beans, served hot; gelatine jellies and custards sweetened with saccharine; nuts of any sort, except chestnuts. Black coffee (claret or whiskey, if desired).

In addition to these foods it is advisable to begin the diet with an addition of a small amount of carbohydrate, 15 to 20 grammes. The reason for this is that certain patients develop quite rapidly a definite acidosis when suddenly deprived of all carbohydrate. A slice of bread, three by four inches, and one-half inch thick, will approximate 10 grammes of starch. Twenty grammes of starch a day, two slices of bread, is adequate protection from serious acidosis. A positive Gerhardt's test may be ignored when the ammonia nitrogen is but 1 or 1.5 grammes per day. On this diet many diabetics cease to excrete glucose within ten days. After the urine has been kept free of

¹ Part of bread allowance may be taken at breakfast.

² The remainder of the day's allowance of bread should be used at this meal.

sugar for several days the diet is to be enlarged by the method to be mentioned later.

On the above diet other cases of diabetes continue to excrete small amounts of sugar after ten days (class 2). As this sugar, even though trivial in amount, indicates that hyperglucæmia still persists, and further dietetic change is required, it becomes necessary to interpolate days when the total quantity of food is restricted. Naunyn used a starvation day, but the same end may be obtained with less discomfort to the patient by a scheme such as the following:

Morning: Omelette of four egg yolks with tomatoes and parsley; 1 large cup of coffee with tablespoonful of cream.

Noon: One small piece of fish, 50 grammes; spinach with butter or oil *ad libitum*; one glass of claret or whiskey.

Four o'clock: Cup of bouillon.

Evening: Asparagus or cabbage served hot with butter; yolks of two eggs soft boiled; tea or coffee (no sugar or cream).

This vegetable day may be used once a week or at most every fourth day. The urine of this day must be watched for signs of acidosis. When the total sugar excretion has been reduced to five or ten grammes a day and will not reduce further, as occasionally happens, this vegetable day is a potent means of clearing up conditions.

Up to this point no mention has been made of the dietary indications in acidosis. When this is a pronounced condition it is necessary to use alkalies at all times and to meet the condition in so far as possible by diet. The oatmeal diet gives the best results, and consists of 64 ounces of oatmeal gruel,³ black coffee in small amounts if desired and water *ad libitum*. This diet is given solely to combat acidosis, but it not infrequently happens that sugar excretion diminishes or vanishes with its use. An oatmeal day may be used once or even twice a week in severe cases, and when the sugar excretion is excessive or obstinate a vegetable day followed by an oatmeal day reduces both sugar and ketone excretion.

³ To prepare oatmeal gruel cook in a double boiler, for at least 6 hours, ten ounces of oatmeal in two quarts of water, slightly salted. While still hot strain through a sieve and add three ounces of butter and stir well.

When the urine becomes free of sugar it is wise to delay at least five days before permitting an increase in the starch ingest. The increase must be made sooner or later if it is possible to do so without inducing a return of glucosuria. In order to facilitate this gradual building up of a diet the unit table is employed. The starch content is expressed in units, instead of grammes, as patients grasp this more quickly; ten grammes of starch is one unit. The values are approximate only. Table II supplements Table I; as all foods in the latter may be used *ad libitum*.

TABLE II.

The food in this list to be taken only in the amounts ordered.

Soups:

Bean	average portion equals one unit.
Clam chowder	average portion equals one unit.
Cream of corn	average portion equals one unit.
Pea purée	average portion equals one unit.
Potato	average portion equals one unit.
Tomato	average portion equals one unit.

Vegetables:

Beans, baked,	2 tablespoonfuls.....	equal 2 units.
Beans, butter,	2 tablespoonfuls.....	equal 1 unit.
Beans, lima,	2 tablespoonfuls.....	equal 2 units.
Beans, kidney,	2 tablespoonfuls.....	equal 2 units.
Beets,	2 tablespoonfuls.....	equal 1 unit.
Corn, green,	1 ear.....	equals 2 units.
Onions,	2 onions.....	equal 1 unit.
Corn, canned,	2 tablespoonfuls.....	equal 2 units.
Green peas,	2 tablespoonfuls.....	equal 1 unit.
Potato, baked,	1 medium sized.....	equals 3 units.
Potato, boiled,	1 medium sized.....	equals 3 units.
Potato, mashed,	2 tablespoonfuls.....	equal 2 units.

Fruit:

Apple,	1 medium sized.....	equals 2 units.
Blackberries,	2 tablespoonfuls.....	equal 1 unit.
Currants,	3 tablespoonfuls.....	equal 1 unit.
Huckleberries,	2 tablespoonfuls.....	equal 1 unit.
Orange,	1 medium sized.....	equals 2 units.
Peach,	1 medium sized.....	equals 1 unit.
Pear,	1 medium sized.....	equals 2 units.
Plum,	2 medium sized.....	equal 1 unit.
Raspberries,	3 tablespoonfuls.....	equal 1 unit.
Strawberries,	4 tablespoonfuls.....	equal 1 unit.

Cereals:

Bread, slice 3 x 4 x $\frac{1}{2}$ inch.....	equals 1 unit.
Hominy, boiled, 1 tablespoonful	equals 1 unit.
H-O, boiled, 2 tablespoonfuls.....	equal 1 unit.
Macaroni, boiled, 2 tablespoonfuls.....	equal 2 units.
Macaroni, baked with cheese, 2 tablespoonfuls.....	equal 2 units.
Oatmeal, boiled, 2 tablespoonfuls.....	equal 1 unit.
Rice, boiled, 1 tablespoonful	equals 2 units.
Shredded wheat biscuit, 1	equals 2 units.
Spaghetti, baked with tomato, 2 tablespoonfuls	equal 2 units.

One may begin by advising for a diabetic whose urine has been free of glucose for one week, that he use three units a day (30 grammes of starch). If there be no return of sugar one unit may be added every week until the patient is using 70 to 80 grammes of starch per day. Further additions should be less frequent and it is a safe rule to permit no more than ten units (100 grammes starch) during the first six months of treatment, even though the urine may be constantly devoid of sugar.

There are undoubtedly cases of diabetes in which the foregoing dietetic treatment will not render the urine sugar free. These require such a very careful balancing between ingestion of starch and excretion of sugar that it can be done only by weighing each, and the consideration of this would lead us away from what we wish to emphasize, that diabetic patients should receive dietetic treatment while under the surgeon's care and that this will favorably influence the surgical condition, and, in the majority of cases, will save the patients from the grievous mutilation of amputation through the thigh. Although arteriosclerosis is usually present, we believe that it is the diabetes which is the unfavorable condition which impairs the cellular nutrition and so lowers cell resistance, rather than the arteriosclerosis, and so these cases demand and must receive a different treatment than those suffering from so-called senile gangrene dependent on endarteritis obliterans.

We wish to express our thanks to Drs. Blake and Peck for the privilege of reporting their cases.

COMPLETE AVULSION OF THE SCALP

WITH A REPORT OF A CASE.*

BY FREDERICK FLAHERTY, M.D.,

OF SYRACUSE, N. Y.,

Professor of Clinical Surgery in Syracuse University.

COMPLETE scalping of an individual is a sufficiently rare accident to make the report of a case of interest. A few scattering cases have been reported in literature until 1910, when Davis, of Baltimore, in an original memoir reported two unpublished cases, one in the service of Dr. Bloodgood and the other his own, together with a rather exhaustive review of the literature upon this subject, reporting in all 91 cases of complete scalping, 80 of which were due to machinery. In 53 cases due to machinery the line of tearing included one or both eyebrows, in 19 cases it passed above the eyebrow, while in 8 cases it was not reported. Another interesting fact was that 79 of the 80 cases were females and one male, and he was a Chinaman and caught his cue. There was periosteal defect in 29 of the cases. Fifty-seven cases were grafted with complete healing in 33 cases. The mortality was 10 per cent. Of the other 11 cases of complete scalping which Davis reported, 7 were done by Indians and 4 occurred from other causes.

The case which I now report is that of a woman, who on September 9, 1912, while working in a private laundry, caught her hair in a revolving shaft. The scalp was completely torn from the skull from a line below each eyebrow in front, including the upper half of the right ear and the upper portion of the left ear, back to the hair border posteriorly. This made a denuded area 13 inches by 16 inches. There was an area of denuded bone over the left side of the frontal and left parietal bone, which measured about 7 inches in length and 2 inches in width.

I saw her soon after she entered St. Joseph's Hospital. She

* Read before the Syracuse Academy of Medicine, October 7, 1913.

FIG. 1.



September 12, 1912. Three days after the accident.

FIG. 2.



July, 1913. Ten months after the accident.

FIG. 3.



July, 1913. Ten months after the accident.

was perfectly conscious, and not suffering much from shock. There was very little bleeding. The interne had tied one small artery on the top of the back of her head. There was avulsion of the right thumb and laceration of the ring finger. To me it was the most frightful appearing trauma I had ever seen in my experience at the hospital.

She stated that she was fixing the washing machine, which was in motion, while making an adjustment she suddenly raised her head. Her hair was immediately caught on a rapidly revolving shaft, winding the hair tightly about the shafting, completely removing the scalp. She put her right hand up to her head in an attempt to free her hair, the hand was caught, the thumb and ring finger were badly lacerated. The patient was alone in the laundry at the time of the accident. As soon as her hair caught she called loudly for help. There was no one at hand so that she was obliged to extricate herself from the machine and walked a distance of twelve feet, where unassisted she stopped the motor. Her hair and scalp were found tightly wound around the shafting. After stopping the motor she sat down in a chair and wrapped a towel around her head. She remained perfectly conscious and was able to give an accurate account of how the accident occurred.

We did not make any effort to cleanse the surface other than to apply hot boric acid compresses. Four days later under ether anaesthesia we covered as much of the right side of the head as was feasible, using Thiersch grafts from the right thigh. Practically every graft we applied at this time grew and became good, firm skin. One week later we covered the left side of the head, using the same method and taking skin from the left thigh. At the second operation we bored a series of rings, using a trephine, through the outer plate of the skull in the area of bare bone, for the purpose of allowing the granulations to grow up and afford a granulating surface on which later we could graft.

On October 28, five weeks later, we found the denuded area covered with healthy granulations. Again with ether anaesthesia we covered this area with Thiersch grafts. This idea occurred to me from a note in an article by Mellish in the *ANNALS OF SURGERY* in 1904 where he quoted a man by the name of Vance, who in 1777 advocated the boring of small holes in the outer plates of denuded bone following scalping by Indians, for the purpose of favoring the healing of the surface by granulation. This proved

to be a very valuable means of covering the bare bone. However, before this area entirely healed, a number of small spiculæ of bone were thrown off, which were the centres of the rings made by the trephine.

In another case I should remove these little plates before placing the grafts. The upper left eyelid at the time of the third operation was retracted and necessitated a graft, to overcome a marked retraction of the eyelid.

Our method of Thiersch grafting is a modification of the regular Thiersch graft, which we have now used for several years, and we find it much more efficient. The grafts are removed in the regular manner, using a sharp razor on the stretched skin. They are then applied to the raw surface, allowing the serum to hold them in place, no solution or moisture being applied. After having covered the desired surface with grafts, it is immediately covered with either bismuth powder or gauze covered with sterilized vaseline, or simple bismuth ointment. This dressing is allowed to remain in place for 4 days, when upon removal, the grafts are found growing much more satisfactorily than when the moist method is used.

Several principles of skin grafting were clearly demonstrated in this case, which I would like to emphasize. First, only autodermic grafts should ever be used to obtain the best results. One so often reads of the self-sacrificing friend or relative, who is placed under an anæsthetic in order to furnish skin so that it can be placed on the injured person. Only about one year ago the newspaper reported the death of one such person, who died while giving his skin to another.

Skin taken from another individual does not grow nearly as satisfactorily as when taken from the individual himself, much less does the skin from animals, etc. In practically all the cases recorded there was either complete or partial failure, and even if the grafts grew, they usually sloughed.

In the case I am now presenting I found it possible to get very good grafts from the same area on the thigh, where I had obtained grafts only 6 weeks previously. All that seemed necessary was not to make the grafts too thick.

In all of the cases reported, great difficulty has been found in healing small areas of ulceration, which later form as the result of excoriation due either to the wig or lying on the head. Our method of treating these small ulcerations, which we found to be very simple and yet very efficient, was to plant one or two small Reverdin grafts in the ulcerated area. This has always produced very quick and satisfactory results.

In a case of complete avulsion reported by Abbe, it was estimated that over 12,000 grafts were placed before a satisfactory result was obtained. In the two cases reported by Davis, the one in Dr. Bloodgood's clinic was admitted August 24, 1906, was discharged March 8, 1907, but three years later there still existed five small ulcers, the largest being as large as a ten-cent piece. The case had been treated by Thiersch grafts, after first allowing granulation to form. The first graft being taken from another person with total failure, later autodermic grafts were successful.

The second case was injured July 29, 1907, six weeks later a Thiersch grafting was attempted with unsatisfactory results. The following year Thiersch grafts from a lamb were unsuccessfully tried. In May, 1909, nearly two years after the accident Dr. Davis, after thorough preparation and treatment of the granulating surface, grafted whole thickness grafts successfully in about eight different operations. He believed that he obtained much better results than occurred in the cases in which the Thiersch method had been employed.

The question naturally arises in these cases whether it is not possible to replace the scalp if the case is seen immediately. Davis found in his series, that 21 attempts had been made to replace the scalp in complete avulsion, with total failure in every case with a possible exception of one, in which the replaced scalp died, but turned into a parchment-like covering, which remained adherent to the cranium, and under which healing took place without complications. Thus it can readily be seen that it acted only as a form of a dressing, and not in any way as a graft.

Robinson, in *Surgery, Gynecology and Obstetrics*, volume

ii, reports a case of complete avulsion in which he used Thiersch, and Reverdin, and later Wolfe-Krause grafts at different times, starting the grafting immediately after the injury, and not waiting for granulation. He, likewise, found the grafts taken from the patient's relatives at the end of 3 or 4 weeks, dissolved or digested notwithstanding that they grew nicely for 2 or 3 weeks and finally disappeared. He agreed with Davis that the whole thickness grafts when autodermic seemed to give the best results.

Contrary to the opinion of many observers, I have never found that simple ointments macerated or tended to destroy the grafts when autodermic, but on the contrary simple sterilized vaseline with or without bismuth has given me most excellent results as a protective covering in many cases.

My case left the hospital in 67 days, with the head completely covered with good firm skin, but like all of the other cases reported there was a tendency for small ulcerations to form for a short period. In the case reported by Binings, of New York, in the *Philadelphia Medical Journal*, June 7, 1902, he had a similar experience in using grafts other than from the individual herself. It would, therefore, seem that in extensive skin grafting certain principles should be observed. First, that it is not necessary for granulation to form in order to obtain the best results, again, that it is useless to attempt to replace large areas of skin completely separated from the body, and that only autodermic grafts should be used, and that it is possible to secure good grafts from the same area if necessary in 5 or 6 week intervals.

INTRATHORACIC GOITRE.

REPORT OF TWO CASES WITH MARKED DISPLACEMENT OF TRACHEA.

BY O. F. LAMSON, M.D.,

OF SEATTLE, WASH.

THESE two cases of intrathoracic goitre which were recently operated on by the writer, seem to be of sufficient importance to warrant a brief report of their clinical progress and ultimate relief through surgery.

The symptoms of these two cases were necessarily chiefly respiratory because of pressure on the trachea, bronchi and lungs, and consisted of coughing, wheezing and dyspnoea which, at times, almost amounted to suffocation.

In the diagnosis, aneurism and malignant growths were considered in both cases, but in the absence of a bruit, thrill and pulsation over the area of dulness, the former was ruled out and malignancy was finally ruled out on account of the chronicity of the symptoms and lack of emaciation.

The history of the onset of the symptoms, together with a circumscribed area of dulness, and the X-ray plates which revealed a tumor beneath the sternum extending up to the lower pole of the thyroid gland, with displacement of trachea to the right, led to the belief that the intrathoracic growths which undoubtedly were present, had their origin in the thyroid gland, and the findings in the operating room confirmed this conclusion.

At the time of their appearance in the office, there was a marked similarity in the symptoms the patients complained of, and examinations revealed similar findings; however, there was a difference in the manner of their development.

In Case I there was a cyst which developed at the lower pole of the gland, as it grew, extended downward into the mediastinum, and at the same time there was some enlargement of the thyroid gland in the cervical region, which about one and a

half years ago disappeared, as the patient stated (probably during a fit of coughing it was drawn beneath the clavicle and was retained in the thoracic cavity); after that, beginning rather acutely, there was a marked increase in the severity of her symptoms which were gradually growing worse.

In Case II there was never any sign of any visible goitre, the growth having developed at the lower pole of the gland and extended downward into the mediastinum, during the entire course of its growth, producing such marked narrowing of the trachea as to almost strangle the patient.

This patient had been treated for asthma off and on for ten years, both by internal medication and at various health resorts, and recently underwent operations on the nasal passages with the idea of relieving his symptoms.

When both of these patients were seen at the office breathing was extremely difficult and both gave histories of spells in which they were threatened with suffocation.

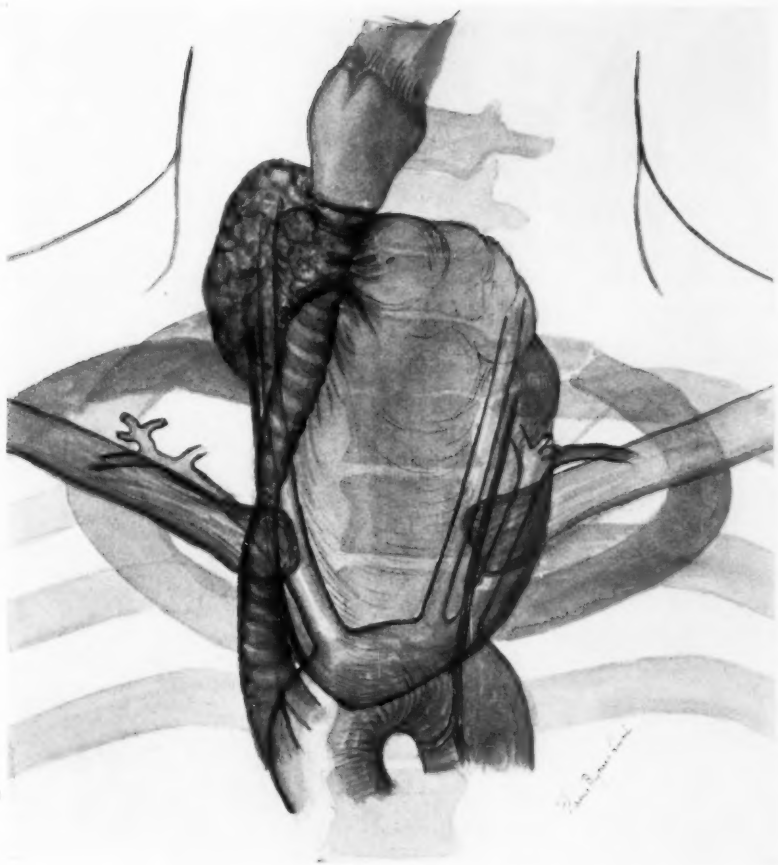
CASE I.—T. V. M., female, age forty-five. Married.

Personal History.—Always been well except for present trouble. Has borne two children, ages sixteen and fourteen respectively.

Present History.—Patient seeks medical aid because of cough and shortness of breath, which has been marked for the past one and a half years and gradually getting worse. Patient says that for several years past she has had some enlargement rather low down, in region of left lobe of thyroid gland; about one and a half years ago the mass disappeared and shortness of breath became marked, from which time it has gradually grown worse; past four weeks dyspnoea has been very distressing and has had six or eight spells in which she almost strangled. Her voice has been husky for past six months.

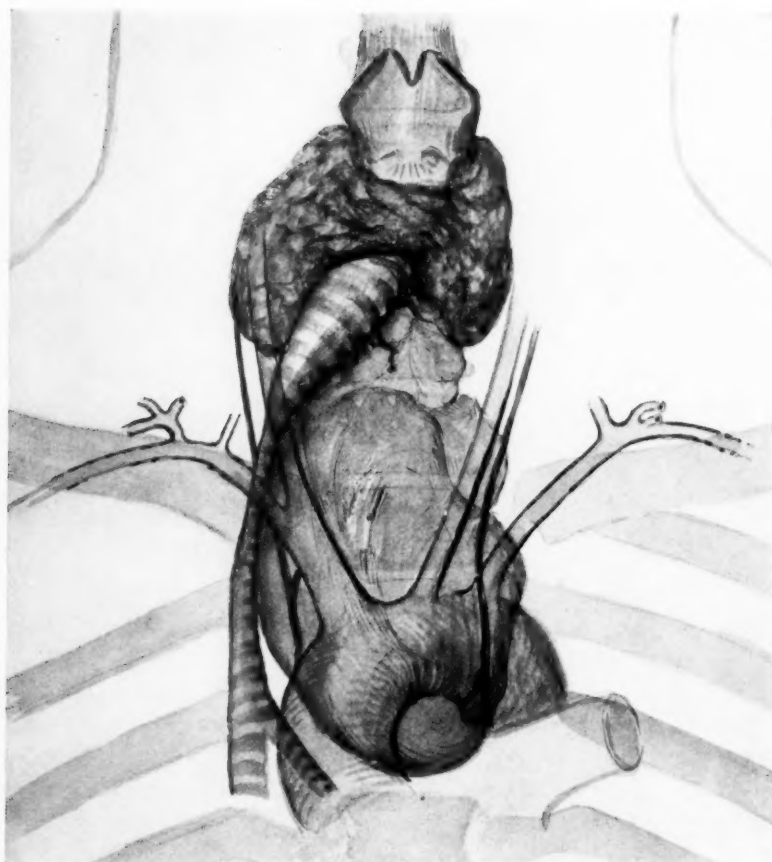
Examination.—No visible tumor on ordinary respiration but on having patient cough there was noticed a rather marked bulging in suprasternal and supraclavicular region. Larynx and upper trachea could be noticed pushed to the right. Percussion revealed an area of dulness extending to right of midline downward to third rib and outward to left of midline about three and one-half inches and upward to the lower pole of left lobe of thyroid gland.

FIG. 1.



Drawing constructed after X-ray plate.

FIG. 2.



Drawing constructed after X-ray plate.

X-ray revealed shadow in region corresponding to the dulness on percussion, and displacement of trachea to right (see Fig. 1, constructed from skiagraph). Examination of throat revealed paralysis of left vocal cords.

Operation (June 3, 1913).—Done under local anæsthetic (novocaine) because of marked dyspnœa and fearing that patient would not take general anæsthetic well. Large colloid cystic goitre (intrathoracic) removed. Patient's breathing improved immediately and she has made an uneventful recovery.

Skiagraph taken two months after operation shows trachea in normal position.

CASE II.—F. N. S., male, age thirty-nine.

Family History.—Mother and her two sisters were afflicted with goitres.

Personal History.—Says he was never seriously ill except for so-called asthmatic spells which have bothered him off and on for ten years.

Present History.—Patient says he has been troubled with asthmatic attacks for about ten years, coming on intermittently and usually preceded by a slight cold, and worse in winter. For past four years troubled with distressed breathing, wheezing, cough; been gradually getting worse and has noticed some enlargement in region of right lobe of thyroid gland (caused by displacement of trachea to right and not by any enlargement of right lobe of gland) for past two years. January, 1913, had a sensation as though he was being strangulated, which persisted for about four weeks, coming on following a slight cold, also a similar spell in June, 1913.

Examination.—Patient's facial expression one of extreme anxiety; breathing is of stertorous type, face flushed (at times slightly cyanotic). Percussion reveals dulness extending from right of midsternal line to left about three and a half inches and downward to third rib and upward to lower pole of right thyroid gland. X-ray revealed shadow in this region, and displacement of trachea to right as in Case I (see Fig. 2, constructed from skiagraph).

Operation (July 3, 1913).—Local anæsthetic (novocaine) for same reason as in Case I. Removal of cyst resulted in marked improvement in patient's breathing before he left the operating table and his recovery since then has been complete.

Skiagraph taken twenty-five days after operation shows trachea in normal position.

The writer wishes to express his appreciation to Dr. Wm. Teepell for the most excellent X-ray plates from which the diagrams were constructed.

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AN ŒSOPHAGOSCOPE WITH DIRECT OUTSIDE ILLUMINATION.

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My object in bringing out this form of œsophagoscope is to have, first, a strong projected illumination with a minimum of light reflexes; second, to have the electrical connections entirely outside the tube; third, to have the electrical connection as simple as possible; and fourth, to have the whole instrument, except the ocular and electric light, sterilizable by boiling. It has also been my object to have a universal light carrier adaptable to any length and size of œsophagoscope (Fig. 1).

Description.—In many details this instrument differs from the others now in use. Its principle may be understood by referring to Fig. 2.

The tube part of the œsophagoscope (Fig. 3) is 48 cm. long and 10 mm. in diameter. At the distal end is the usual ring placed to guard against wounding the tissues during introduction (it is entered in the œsophagus over a flexible bougie). At the proximal end of the tube is an enlargement 20 mm. in diameter tapered on the outside to fit the light carrier, and conically bored on the inside eccentrically to the axis. This whole proximal end piece is attached to the tube eccentrically. The light carrier is shown in Figs. 4 and 5. It consists of a cylinder 52 mm. in length, with an internal diameter of 20 to 22 mm., to fit over the end of the tube. Let into one side of it is a half cylindrical chamber 25 mm. in length and 8 mm. in diameter. In the distal end of this chamber is a planoconvex lens. On the opposite side of the light carrier is a small pet-cock permitting the introduction of air under pressure when needed. At the proximal end of the light carrier is fitted an air-tight eye piece containing either a plain glass or a lens, as may be required. This is removable when it is desired to pass instruments down the tube. The light is supplied by a high efficiency electric bulb, which is set into the hemicylindrical chamber from the outside. This light can be moved backward or forward by loosening the thumb-screw (*A*), thereby focussing the light with a certain degree of accuracy at the end of the long tube. The divergent rays are cut down by a small diaphragm which encircles the 8 mm. lens (*B*). The object in this way of dealing with the light problem has been to utilize one-half of a strong electric light, and to throw the rays by means of the planoconvex lens down the tube as parallel rays. At the same time, the

polished interior of the tube aids in the lighting effect. The amount of the encroachment of the hemicylindrical chamber which holds the light (*C*) upon the lumen of the tube is regulated by turning the light carrier upon the eccentrically placed proximal end of the tube. It is possible in this way to get ample illumination and, at the same time, make use of instrumentation through the tube without any interference from the hemicylindrical chamber of the light carrier. When the eye is applied to the eye piece, the full field at the end of the tube can be seen without interference to vision.

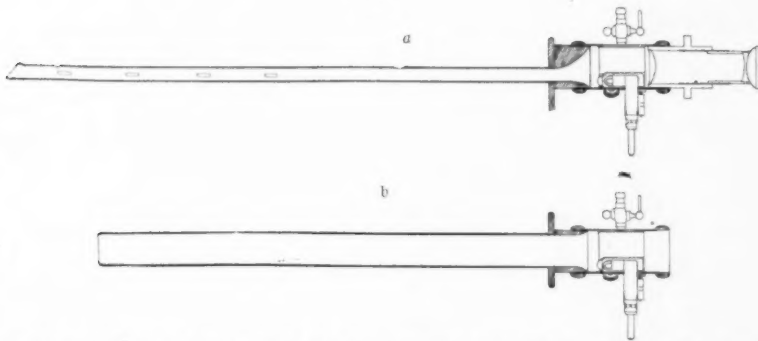
I have been fortunate in having the advice and assistance of Mr. R. Wappler in working out the mechanical details of this principle. The instrument has been in use for a year and has given satisfaction.

FIG. 1.



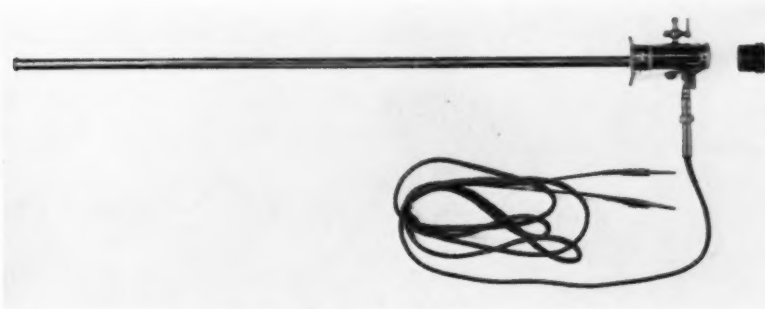
Showing *a*, gastroscope; *b*, oesophagoscope; *c*, bronchoscope; *d*, proctoscope tubes; *e*, universal light carrier.

FIG. 2.



Showing *a*, section of bronchoscope with lens system; *b*, section of proctoscope. It is to be observed that the light carrier fits either tube, although they differ greatly in calibre.

FIG. 3.



Showing oesophagoscope assembled.

FIG. 4.

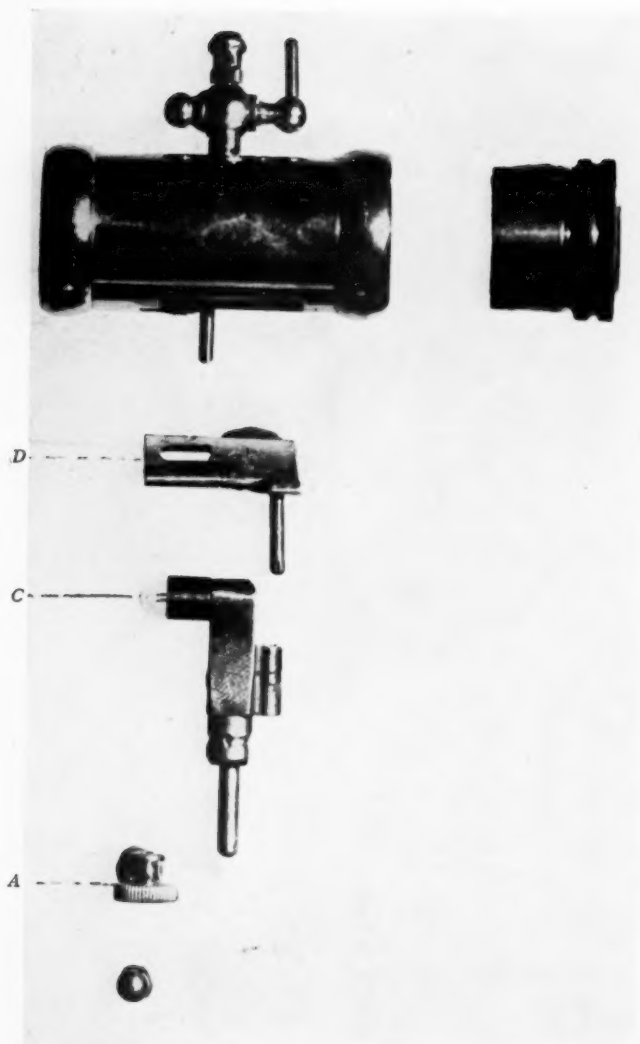
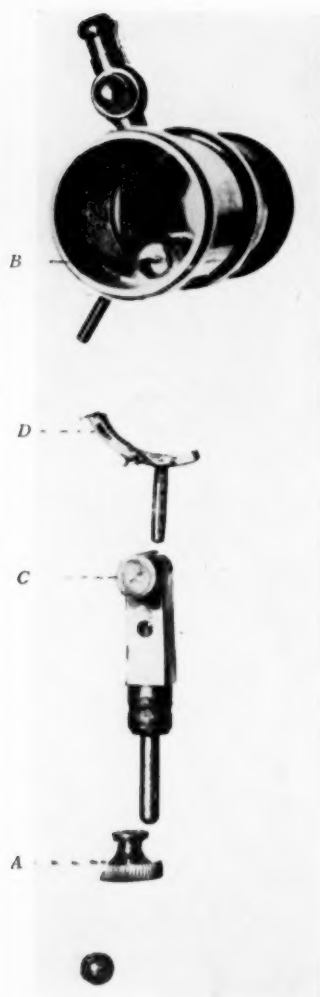


FIG. 5.



FIGS. 4 and 5.—Showing component parts of light carrier. Thumbscrew A, for focussing slide D, to which is attached electric lamp C. The focussing is accomplished by moving the lamp toward or away from the small planoconvex lens B.

THE RELATION OF POSTERIOR SUBLUXATION OF THE SHOULDER-JOINT TO OBSTETRICAL PALSY OF THE UPPER EXTREMITY.*

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OBSTETRICAL palsy involving the upper extremity only is ascribed almost universally to injury of the brachial plexus affecting, especially, its fifth and sixth cervical roots. It is almost as generally agreed that the much more frequent brachial palsies from injuries of the shoulder region in adults, are due to a similar lesion of the brachial plexus. In a study of the latter, published in the *ANNALS OF SURGERY*, January, 1911, I concluded that most of them resulted from sprains and dislocations of the shoulder-joint, the essential lesion in both being a laceration of the axillary portion of the joint capsule and the palsy resulting from the inclusion of the branches of the brachial plexus in the adjacent axillary inflammation. Because the obstetrical palsies are, evidently, of the same nature as these adult palsies, I suggested that in many cases they also are due, primarily, to injuries of the shoulder-joint. In June, 1912, in Nos. 23 and 26 of the *Münchener Medizinische Wochenschrift*, F. Lange published two papers, in which he put forth, essentially the same theory. In the first paper he discussed under the title of "Distortion of the Shoulder" which he ascribed to laceration of the anterior portion of the shoulder capsule, the same group of cases, evidently, which I had described as "Stiff and Painful Shoulders,"¹ and had ascribed to laceration of the antero-inferior portion of the capsule. According to my experience and judgment, all sprains and dislocations of the shoulder-joint are associated early with some brachial palsy, as well as with stiffness and pain at

* Read before the Philadelphia Academy of Surgery, October 6, 1913.

¹ American Journal of the Medical Sciences, April, 1911.

the shoulder. In most cases the palsy is mild and transitory, in some it is very severe, but even in these it is probably rarely permanent. Lange did not refer to the palsy in his adult cases but in his second paper, devoted to obstetrical palsies of the upper extremity, he ascribed most of these palsies to laceration of the capsule of the shoulder-joint. Of his 17 palsied arms (in 15 patients), he regarded 13 as undoubtedly pseudopalsies, due to laceration of the capsule, and in 15 of the 17, he found the same position of the arm as in his "distortions" of the shoulder in adults. According to Lange, Küstner believes that the obstetrical palsies are due to epiphyseal separations of the upper end of the humerus with rotatory deformity, while Finck believes that they are due to preglenoid dislocations of the shoulder.

While the brachial plexus theory is generally accepted, particularly for the obstetrical palsies, there has been much discussion as to how the plexus is injured at birth and a variety of mechanisms have been suggested. The greatest difficulty has been found in accounting for the localization of the injury to the junction of the fifth and sixth cervical roots of the plexus. When a more or less general brachial paralysis follows soon after an injury to the shoulder region, the obvious cause is an injury to the brachial plexus. The relation of an injury of the shoulder-joint to such a paralysis, is much less obvious. It has seemed to me that the localization of the injury to the junction of the fifth and sixth cervical roots rested on an uncertain basis until supported by operations on the plexus. Certain it is that in the great majority of cases, electrical examinations are not made to determine what muscles are paralyzed. The diagnosis is, evidently, made upon the history of a paralyzed upper extremity, first observed immediately after birth, and upon the characteristic internal rotation of the whole limb. Electrical examinations soon after birth are exceedingly unsatisfactory and unreliable and are rarely made. Fairbank,² who recently reported probably the largest experi-

² *Lancet*, Lond., May 3, 1913, p. 1217.

ence with obstetrical palsy, says that electrical examinations are not advisable before the end of the second month, the use of an anæsthetic being essential, but by this time the case will probably show definite signs of recovery, so as to render electrical examination unnecessary. Sherren does not test the electrical reactions until the age of three months.³ The fact that, in most cases, there is practically no disturbance of sensation in the affected limb, although the roots of the brachial plexus are all mixed nerves, has not been satisfactorily explained.

The most substantial support of a plexus origin has been furnished, in about the last decade, by a few surgeons who have exposed the plexus in a small number of cases. In most of them the plexus was found enveloped in adhesions, in some the nerves were thickened and in a few one or more roots were found divided and the torn ends retracted. Only the last group, in my opinion, furnish substantial evidence in favor of the plexus theory, and even these need further confirmation. Without operation, it appears that the paralysis recovers spontaneously, in most cases, after a time, and time is not particularly important to these young patients. It has seemed to me that before the plexus should be exposed, definite areas of impaired sensation should be located. If these operations become common, there will probably be fewer complete recoveries of the paralyses than there are now. The operation was advised in one of my cases with posterior subluxation and was refused. That patient has been improving ever since and after about three years has recovered much power in the affected limb. In my opinion, the average surgeon without the facilities for careful study by dissection of the anatomy of this region, should not attempt to lay bare the delicate cords of the brachial plexus in the young child. The plexus is deeply situated in a confined region, surrounded by very important vessels and nerves, the cords of the plexus lying close together interweaving with each other and embedded in a mass of adhe-

³ *Injuries of Nerves and Their Treatment*, 1908, p. 209.

sions. Even in the hands of the able surgeons who have reported the results of their operations the proportion of cases showing divided nerve roots has been very small. Fairbank operated on five cases and found rupture of the nerves in only one. This involved the fifth and sixth cervical roots which were torn across at their junction. Even in this case electrical stimulation before operation and of the nerves at operation, "made it certain that there must have been some fibrils passing on from the fifth cervical root or the fifth and sixth cervical to the bulbous common trunk (distal end of rupture), though dissection suggested complete division." Lange exposed the branches of the brachial plexus in the axilla in a case of obstetrical palsy, and found the cause of the paralysis to be the embedding of the nerves in thick connective tissue, for an extent of 4 cm., and found also a diminution and deformation of the head of the humerus. This is the only case of obstetrical palsy, of which I have knowledge, in which the nerves have been exposed in the axilla where they are adjacent to the shoulder-joint. I would suggest that the exudate and adhesions about the plexus, found so uniformly in operations above the clavicle, are the result of extension upward of the blood and synovial fluid from an injured shoulder-joint, which in the new-born is only a few inches below the plexus. The almost recumbent position of the infant would favor this upward extension. Boyer⁴ recently reported a very interesting case of obstetrical palsy in a woman who died in an insane asylum, and upon whom an autopsy showed clear evidence of tearing of the cervical roots, on the right side, from the spinal cord, the rupture affecting particularly the seventh cervical. The brachial plexus of the left side appeared normal in size and origin. The roots of the fifth, sixth, seventh and eighth cervical on the right side were much smaller than natural and were reduced to fibrous cords, which were impossible of good dissection owing to the abundance of tough fibrous tissue. Boyer calls attention to the fact that the usual excision of a

⁴Proc. Roy. Soc. Med. (Neurological Section), 1912, p. 31.

part of a nerve trunk, as Kennedy suggests, would not have done good in this case but would have done harm. Mills⁵ reported similar findings upon exposure of the cervical portion of the spinal cord by Frazier, in a case of severe brachial paralysis in an adult following an injury to the shoulder region. These cases show conclusively that rupture of the cervical roots of the brachial plexus do occur in some cases, but they do not favor the prevailing view that the rupture usually takes place in the neck at the junction of the fifth and sixth cervical.

My conception of the obstetrical palsies resulted from a previous study of the adult brachial palsies, which in turn had its origin in a study of the anterior dislocation of the shoulder, including its mechanism, lesions and sequelæ. In my judgment, the dislocation is the key to the solution of the problem involved in most of the adult cases. I believe now that the posterior subluxations of the shoulder associated with obstetrical palsies, will prove to be the key to the solution of the problem in most of these cases. Since the adult palsies have led me to study the birth palsies, I wish to state very briefly, my position in connection with the adult cases. The most damaging movement at the shoulder is hyperabduction. Forced external rotation has a similar effect, but is probably of secondary importance to hyperabduction. The capsule maintains the continuity of the skeleton at the joint and takes up, here, the forces applied to the skeleton, so that a break in the skeleton at the joint involves, primarily, the capsule. The end result of hyperabduction at the shoulder-joint is an anterior dislocation, a sprain usually being merely an aborted dislocation. If the joint is immobilized after a sprain or the reduction of a dislocation, a more or less severe palsy of the whole limb will develop because of the involvement of the branches of the brachial plexus in the adjacent axillary inflammation. The palsy and atrophy will be most marked about the shoulder, but occasionally the muscles of the hand and forearm will be most affected, as in Case II. In many cases the palsy improves so

⁵ Pennsylvania Medical Journal, 1910-1911, p. 850.

rapidly that it is overlooked or ignored. Sometimes it is so severe and persistent that it cannot be ignored and then is usually ascribed to an injury of the brachial plexus. The best evidence that it is not due to an injury of the plexus is the fact that with the restoration of the normal motion to the shoulder-joint the palsy disappears rapidly. If the dislocation remains unreduced, the palsy will improve slowly, but will never entirely disappear because of the interference with the function of the joint, and perhaps also because of pressure of the dislocated head on the adjacent nerves. I have selected the following adult cases for illustration of different types of brachial paralysis of shoulder-joint origin:

CASE I.—A woman, forty years of age, teacher of drawing. On May 13, 1913, on stepping from a row-boat which began to move away from the shore of a small lake, she grasped a post on the shore with her right hand, keeping both feet in the boat. The boat moved from the shore and dragged her feet until her body was almost horizontal and her right arm in full abduction, in which position she pulled the boat to shore again. She had some pain in the shoulder immediately but it was not particularly noticeable until about an hour later, and on the following day it was worse. On the third day Dr. W. Drummond located severe tenderness over the greater tuberosity of the humerus. On the same evening he manipulated the shoulder to exclude the possibility of a dislocation, and during the manipulations observed a sensation as though the humeral head jumped out of the socket and back again, and felt distinct crepitus. The skiagraph taken on the fourth day seemed to show a fracture of the greater tuberosity, although this was not very clear. The arm was then bound in the Velpeau position, for 14 days, and on removing the bandage, the right arm hung helpless at her side and she could scarcely move a finger. This frightened her very much, because she had recently come from England to take a position as teacher of free-hand drawing, which required perfect movement of the arm. She was compelled to cancel her engagement and go back home. An insurance company, after the examination by its physician, quickly settled with the patient on a basis of five months' incapacity for work, indicating that the insurance physician regarded the

paralysis in a serious light. In the three days which intervened between the removal of the bandages and her departure from the country, there was a considerable improvement in motion and power, especially in the hand and forearm. I had to be content with giving her instruction as to the exercises which she should follow. I heard first from her under date of August 12. She still suffered from pains in the shoulder and arm, but the power and movement of the limb were "wonderfully better." She was still unable to put her hand back far enough to fasten a dress skirt, could not put the hand to the back of her head without pain, and could not raise the arm straight above her head. These, however, are among the last restrictions of movement to disappear. She had fitted up a black-board recently to try her drawing ability and was "quite greatly surprised as to the power of the right hand." All she needs is to apply force enough to stretch the still contracted tissues sufficiently to give her normal motion.

If the paralysis in this case were due to actual rupture of nerve fibres of the brachial plexus, it should have come on immediately after the injury. That the shoulder-joint was the chief seat of injury was evident from the pain and limitation of movement there, and the joint injury with the associated involvement of the branches of the plexus in the adjacent axillary inflammation, will explain every phase of this case. This type is very common, the palsy affecting most and being last to disappear from the muscles of the shoulder region.

CASE II.—Man, thirty-nine years of age, mechanic. On about May 18, 1911, received a heavy blow on his left shoulder from a falling board, while at his work. Suffered severe pain in the shoulder at the time, and during the rest of the day had a peculiar numb sensation in his left hand but continued at his work. On the following day he had no power in it and the power in his forearm and arm were much below normal. Admitted to the nervous ward of the Philadelphia Hospital, May 20, 1911, in the service of Dr. J. Hendrie Lloyd. The following is a very brief synopsis of Dr. Lloyd's examination of the case: Restricted power in arm and forearm and total paralysis of hand. Has no use of any of his fingers. Complete wrist drop. Ribbon shaped area of anæsthesia, about two inches wide, along ulnar side of forearm.

Scapulohumeral ankylosis. Cannot abduct arm to a right angle. Diagnosis: Musculospiral and ulnar paralysis with total paralysis of hand. After seeing the case with Dr. Lloyd, June 6-19, 1911, he asked me to treat it. On the following day I forced the arm into full abduction and external rotation, with the patient under ether, and fixed it in external rotation and almost full abduction on an obtuse angle splint, keeping the patient in bed. Although I cannot explain the rapidity with which the improvement developed, it was striking. On the following day, June 21, the little and ring fingers could be flexed slightly. June 22, could flex all the fingers slightly. With palm turned downward, could extend hand at wrist slightly. The ribbon-shaped ulnar area of anæsthesia had disappeared to such an extent that he could feel pain sense readily, although he still had numb sensations in this area. June 23, could flex and extend thumb fairly well and could flex fingers about one-fourth way toward making a fist. Motion at wrist also increasing. June 27, could grasp objects weakly with affected hand. Still had slight numb sensations along ulnar border of forearm. July 19, considerable atrophy of left arm and forearm. Sensation good in all parts of limb. Movement in left thumb almost as free as in right thumb, but power much less, and last phalanx could not be flexed as far as in right thumb. Could close fingers about three-fourths as well as on right side. Index finger did not flex quite as well as others. Could approximate thumb to all fingers except little finger. Could close fingers best with hand in dorsal flexion at wrist. Could flex and extend wrist almost as well as on right side, either with palm turned upward or downward. Rotation of forearm about as free on left as right side, although the power was much less. Active flexion and extension of elbow almost as free on left as right side but muscles much weaker. Active abduction at the shoulder to about 100 degrees, passive abduction to about 160 degrees.

The patient returned to work August 28, his work consisting chiefly in chipping iron and steel with a hammer and chisel, the latter being held in the affected hand. At first he had much difficulty in holding the chisel firmly enough, and could not have continued at his work if the foreman had not encouraged him to do so. In about two weeks he could do his work satisfactorily enough. The improvement in the whole limb continued, until in about a

year it was practically normal. He says that for a long time the limb has been about as good as it ever was.

In my opinion, the original injury resulted from forced abduction of the arm at the shoulder, with a laceration of the axillary portion of the capsule, perhaps with a temporary dislocation and immediate reduction as the arm fell to the side. The reparative inflammation extended to the axilla involving the branches of the brachial plexus, and was followed by a contraction of the capsule which accounted for the limitation of abduction and external rotation. The tearing of the contracted capsule and the abducted position of the arm, in some way, were responsible for the rapid improvement which followed, and the exercise of the muscles involved in using the improved motion, which became normal, was chiefly responsible for the return of the power to the normal.

CASE III.—Woman, forty-one years of age, weight last spring 130 pounds, now (September, 1911) 112 pounds. Referred by Dr. B. F. Stahl, who suspects a mild tuberculous lesion in the left lung. No tubercle bacilli in the sputum. In December, 1909, she had a large abscess in the left axilla, which was opened and required about four months to heal. Ever since she has had a severe brachial palsy of the whole limb, most marked in the shoulder region, and marked limitation of abduction and external rotation. Muscles of the whole limb are much atrophied, particularly those about the shoulder, the deltoid being so thin that the upper end of the humerus seems almost subcutaneous. In January, 1911, an effort was made to restore motion to the shoulder by "breaking up adhesions" under ether. Passive movements by a masseuse were continued for a long time but nothing was gained. In November of the same year, I tried the same procedure, but fixed the arm in full abduction for 18 days, after which passive motion and massage were kept up faithfully for about seven months. No improvement in movement or power resulted. Electrical examination by Dr. J. W. McConnell, showed that all muscles of the limb responded to the faradic current. I still believed that if I could restore the normal motion to the shoulder, the patient would recover much of her former power, and I proposed a plastic operation on the scar tissue in the axilla to overcome its effect in restrict-

ing motion. She returned in October asking for the operation, which was performed at the Howard Hospital, November 14, 1912. The arm was fixed at a right angle on a splint for three weeks. Soon afterward she began mechanical treatment in the orthopædic gymnasium of the University Hospital, for which privilege I am indebted to Professor G. G. Davis. This treatment was continued until June, 1913, when she left for her summer vacation. She could then raise her arm straight above her head, by anterior elevation but could not raise it as far by lateral elevation. The improvement in power was general in the whole limb and was still continuing. She could play on the piano, could swing light Indian clubs, and do many other things that she had not been able to do since before the axillary abscess had developed.

As I view this case, the axillary abscess resulted in essentially the same condition as the shoulder-joint injury in the preceding cases, except that in this one the involvement of the branches of the brachial plexus in the scar tissue produced a more severe and unyielding condition than is usual after the joint injury. The release of the nerves from this dense scar tissue must be a slow process. How much return of power will ultimately occur, remains to be seen.

Posterior subluxations of the shoulder-joint associated with obstetrical palsies are only now beginning to receive the attention they deserve. Fairbank says, "Anatomically, there is no doubt about the subluxation." He has notes of 40 cases of obstetrical palsy seen during the last few years, 35 of them in the "last three years or so," and of the subluxation he says it "has received little or no attention in this country" (England). Excluding three of his 40 cases, seen before the subluxations were appreciated, 28 of the remaining 37, or 76 per cent., showed subluxation of varying degree. Nine out of my 12 cases, or 75 per cent., showed subluxation. That they have received little attention is to be accounted for by the fact that they are peculiarly obscure (see Figs. 1, 2 and 3). I had never had any particular interest in obstetrical palsies until I realized that they were generally supposed to be due to the same cause as the

adult brachial palsies from injury to the shoulder region. I then sought a case for study and this was furnished me by the kindness of Dr. R. H. McCombs. I approached the case with the thought of its having, possibly, a shoulder-joint origin. Although a peculiarity in the conformation of the shoulder immediately attracted my attention, it was some time before I realized that the patient had a posterior subluxation of the shoulder. I also realized, at that time, that there was something peculiar about the anterior part of the shoulder, the real nature and significance of which I could not interpret. From my study of this case, as already reported, I concluded that the dislocation was probably the cause of the palsy or pseudopalsy. The cases which I have since seen, have only confirmed that view. In seeking important papers, I did not look up congenital dislocations of the shoulder and for that reason overlooked Whitman's very important paper on this subject.⁶ He called attention to the frequent association of these congenital dislocations with obstetrical palsies. He regarded most of them as being secondary to the paralysis resulting from an injury to the brachial plexus. Fairbank supports Whitman's view. F. Lange who supports the shoulder-joint origin of most obstetrical palsies, disputed the existence of dislocation in these cases.

The theory that these palsies are due, uniformly, to rupture of fibres of the brachial plexus had its origin and became firmly established without taking into account these frequent subluxations. I cannot agree with Whitman and Fairbank that the dislocation is due to the paralysis, but prefer to believe that the paralysis is due to the dislocation, and that the term, pseudopalsy, applied to most of his cases of obstetrical palsy, by Lange, serves a useful purpose. If this can be proved, then it follows that if the dislocation is recognized and completely reduced early enough, in most cases there will be a complete return of function and growth of the affected limb. The chief responsibility will then lie with the obstetrician and family physician.

⁶ ANNALS OF SURGERY, 1905, xlv, p. 110.

To prove this the essential thing is to show that the dislocation occurs at birth and is traumatic in origin. While it is possible to have a dislocation and a rupture of the brachial plexus occur at the same time, this is not likely or, at least, is not likely to be frequent. We have no positive evidence that the sublaxations are due to paralysis. Stimson, like Whitman, included under congenital dislocations of the shoulder, all dislocations present at birth or developing as the result of injury to the brachial plexus, and, like Whitman, regarded the true congenital dislocations as rare. But while Whitman considered those secondary to an injury to the brachial plexus, as the most frequent, Stimson⁷ thought it probable that the most frequent variety was due to force applied to the shoulder at birth. He had 5 cases, all backward dislocations; four of them, possibly all, occurred at birth. Both Whitman and Fairbank say that the shoulder-joint is injured at birth in some cases, and Fairbank states that it would be impossible to say that the dislocation did not occur, in some cases, coincidently with the plexus injury at birth. In one of his cases, the physician who was present at the birth of the patient, diagnosed the dislocation of the shoulder at that time, but Fairbank concluded that the physician must have been mistaken. In 5 of his 28 cases with sublaxation, he completely reduced the dislocation without operation. The earliest period at which he has seen a reducible sublaxation was 2 months. The dislocation in this case had probably existed some time previous to the reduction, which brings its origin close to birth. Of the 28 cases, 14 were in the first year of life. In 18 of the 28, electrical examination, under an anæsthetic, showed no signs of paralysis. Of the 10 remaining, 7 showed only some weakness of the extensors of the back of the hand. It will be seen, therefore, that in at least four of his cases, the paralysis had entirely disappeared within the first year of life, and in 11 it had almost entirely disappeared. A paralysis which disappeared so soon, could hardly be expected to produce, secondarily, a sublaxation of the shoulder that would be irreducible without operation.

⁷ *Fractures and Dislocations*, 1907, p. 610.

I have had only nine cases of obstetrical palsy with subluxation which, I confess, is a small experience upon which to base a dispute concerning the obscure etiology of these cases. I believe, however, that there is in every one of these nine shoulders positive evidence that the subluxations developed at birth from direct pressure against the anterior portion of the shoulder pushing the humeral head backward. It should be borne in mind that these subluxations have been, practically, overlooked until recently. Close observations of them have not yet been reported. In my second case the subluxation was easily recognized although it was of milder grade than in the first, but as in the first I was conscious of the fact that there was something peculiar in the conformation of the shoulder which I did not understand. I was imbued with the idea that all that was necessary was to reduce the dislocation and from my operative experience in the first case, I was convinced that the chief obstacle was connected with the alteration in the glenoid cavity and with the anterior portion of the capsule. I met these conditions in operation on this second case by dividing completely the anterior portion of the capsule and removing the upper anterior margin of the glenoid cavity. Although I restored much of the restricted abduction and external rotation, and the function and development later improved remarkably, I did not completely reduce the dislocation. This became more obvious in the after-treatment when I could palpate the shoulder freely without pain to the patient, and it was then that I discovered what I believe will throw a new light on the subject of obstetrical palsy. If the examining finger is passed from behind over the upper surface of the acromion, on the normal side, it will come upon the rounded upper end of the humerus just in front of the anterior edge of the acromion. By the same manœuvre on the affected side, the finger will not find the smoothly rounded upper end of the humerus, just in front of the acromion. What I had not recognized before was that the anterior portion of the acromion was bent downward and as the finger passed forward it continued in contact with this portion of the acromion a slight distance

downward (see Fig. 1 *a* (*A*), and Fig. 4 (*A*). This change in the shape of the acromion was present in varying degree in all nine of my cases with posterior subluxation.

My only purpose at present is to call attention to its presence and to its probable significance. I am not prepared to describe the deformity in detail because its characteristics vary. This is probably due to the fact that the pressure which is responsible for it is not always applied exactly in the same place. The most important variation was found in my last case, in which the posterior dislocation was more marked than usual. The downward projection of the anterior portion of the acromion was slight or absent and merely changed the normal inclination of the acromion from the anterior margin downward and backward, so that it was horizontal or slightly curved from before backward, which showed that the anterior portion had sustained pressure from above. In this case, however, there is evidence that the coracoid process had received considerable pressure. Efforts to show the deformity by the X-ray have not been satisfactory (see Figs. 5, 6, and 7) ⁸. This may be due in some cases to the fact that the abnormal portion of the acromion is cartilaginous as shown by operation in two cases, and in others to the difficulty in bringing out by the X-ray the difference between the normal inclination of the acromion and the change produced by the pressure. As one would expect

⁸ The downward projection of the acromion which is easily palpable and even visible in Case I—Fig. 1 *a* (*A*) and Fig. 4 (*A*)—is not shown in the skiagraph (Fig. 5, right shoulder). In Case II it is easily palpable and its borders can be outlined, yet it scarcely shows in the skiagraph, Fig. 6 (right shoulder). In Case VIII it was not present although the acromion seemed depressed as a whole and was more horizontal than on the normal side. There is shown in all three cases, by the X-ray, a change in the plane of the acromion. On the normal side it is seen as a thick plate of bone, due to its inclination downward and backward from its anterior to its posterior border. On the affected side, in all three cases, it appears to be thinner, indicating that it occupies a more horizontal plane, which is to be explained by a downward pressure on the higher anterior portion, during birth. Figure 5, right shoulder, seems to show that the humeral head and acromion have been largely worn away, by rubbing against each other during movement. In the normal or left shoulders, the lower margin of the shadow of the acromion marks the posterior margin. This is not so

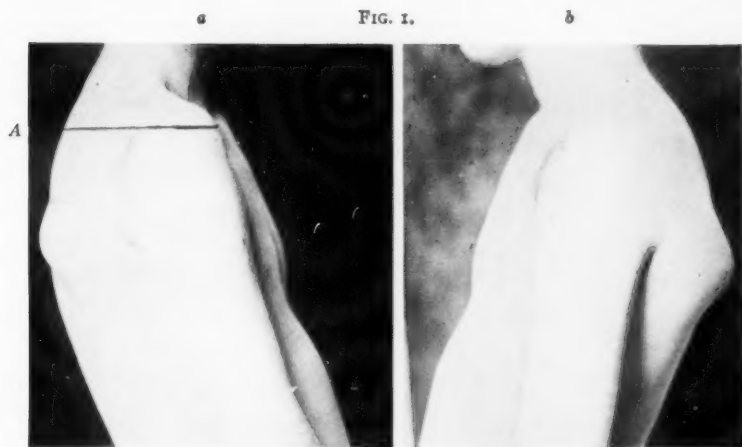
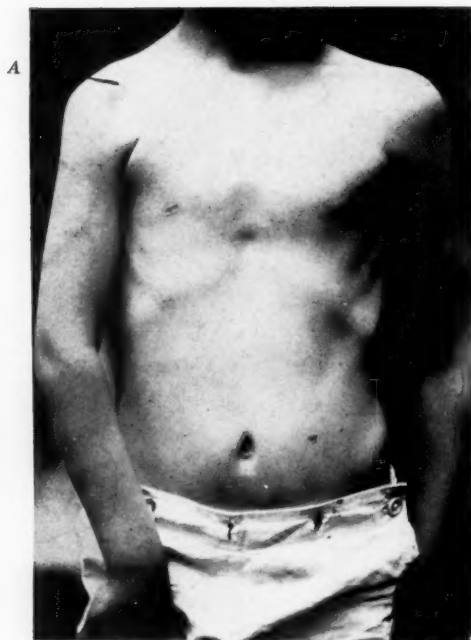


Fig. 1 (Case I), Fig. 2 (Case II), and Fig. 3 (Case VIII) illustrate the mild grade of posterior subluxation in the right shoulder, which is shown by the slight flatness anteriorly. Fig. 1a, A shows the turning down anteriorly of the acromion. The acromioclavicular joint is obviously involved in this case. It is the only case in which the bending down of acromion can be seen.

FIG. 4



Case I. Comparison between the two shoulders discloses a flatness anteriorly in the right one. The turning downward of the acromion is faintly discernible at A. In the other cases careful palpation was necessary to detect it. In this case there is a bony protuberance at the acromioclavicular joint which accounts for the diagnosis of fracture of the clavicle at birth.

FIG. 5a.



Case I. Right shoulder.

FIG. 5b.



Case I. Left shoulder.

FIG. 6a.



Case II. Right shoulder.

FIG. 6b.



Case II. Left shoulder.

FIG. 7a.



Case VIII. Right shoulder.

FIG. 7b.



Case VIII. Left shoulder.

FIG. 8.

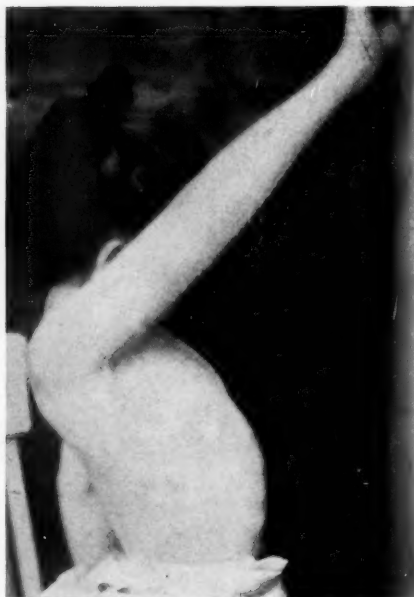
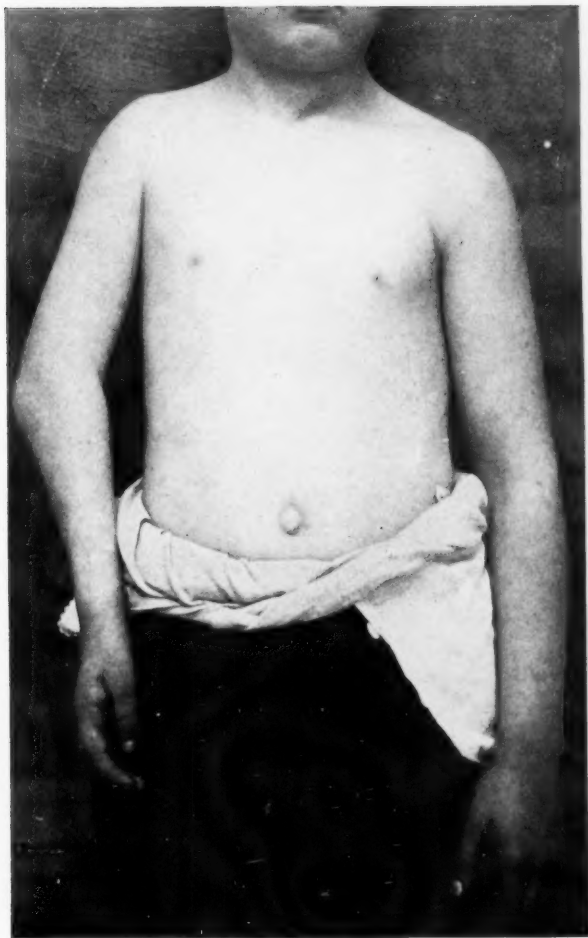


FIG. 9.



Figs. 8 and 9 show degree of active elevation of arm. Mechanical obstruction and not lack of power prevents greater elevation. Patient shown in Fig. 8 has the better development and use of the limb.

FIG. 10.



CASE III. Posterior subluxation at shoulder and anterior dislocation of radius at elbow. Marked compensatory hypertrophy of whole left limb, probably due to unusual helplessness of affected limb.

FIG. 11.



Case VI. Shows limitation of extension at the right elbow, which is about the same in Case IV and in one case without subluxation of the shoulder. Comparison with the other arm shows the internal rotation.

FIG. 12.



Case II.

FIG. 13.



Case V.

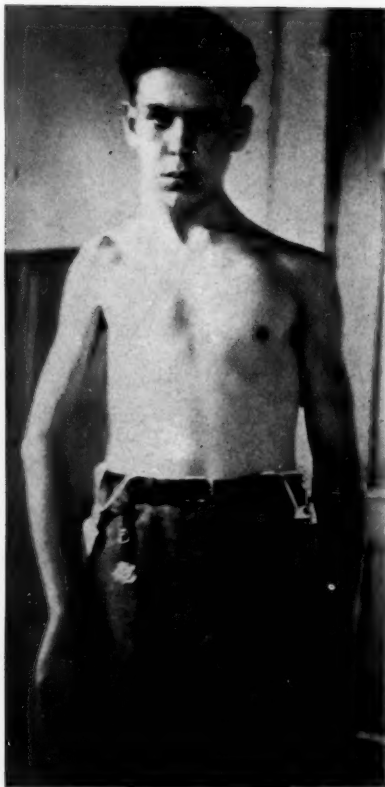
FIGS. 12-14.—Three different positions in which the limb was dressed after operation.

FIG. 14.



Case VIII.

FIG. 15.



Case VIII. Atrophy of whole limb marked, of shoulder and arm muscles more marked than in any of the other cases. The higher of the two prominences seen anteriorly is the edge of the acromion, the lower the coracoid process.

from the fact that the deformity has been overlooked, it is an obscure one. A careful examination is necessary to establish its presence and outlines. The downward projection of the acromion seems almost to fuse with the upper end of the humerus and it is a little difficult to determine when the examining finger is on this portion of the acromion and when on the humerus. It is most evident in the older patients and in them seems to be ossified.

The explanation for it seems obvious. For my cases, at least, it offers positive evidence for what Stimson had already suggested, *i.e.*, that most of the congenital dislocations of the shoulder are probably the result of direct pressure backward on the humeral head by the bony wall of the maternal pelvis, during birth. He says that some of the paralytic forms have been described as "obstetrical paralyses." He also says: "In my four cases, Scudder's two, and Cumston's, the right arm was affected, in Gaillard's the left; and it seemed possible that as the right shoulder is in front in the great majority of births, the cause might be its pressure against the arch of the pubis. Against this or, at least, limiting it, is the double dislocation in Küstner's and the breech presentation in one of mine." My cases furnish a striking confirmation of Stimson's observation. In 11 out of my 12 obstetrical palsies, the right arm is involved.

certain on the affected side. If on this side the lower margin in the skiagraph represented the anterior border, it would mean that this has been bent downward.

A mild downward displacement of the humerus is shown in the three cases. The posterior displacement cannot be shown by this exposure and only with difficulty by any other. Note the absence of the separation between the clavicle and acromion on the affected sides. Figure 5 (right shoulder) seems to show complete fusion of the two bones. There is a bony protuberance at the site of this joint in this patient. The same union of the two bones seems to be shown in Figure 6. In Figure 7, the outer end of the clavicle can be faintly traced, indicating that bony union is not present in this case. The X-ray shows clearly that these are not cases of epiphyseal separation. The small range of movement in the affected shoulder of Case VIII might account for the nearly normal shape of the humeral head (Fig. 7), and probably accounts for much of the atrophy of the muscles (see Fig. 15).

Fairbank, however, says of his cases that "The two arms were affected in an equal number of cases."

The dislocations of the shoulder in the adult are almost always anterior, those associated with obstetrical palsy are practically always posterior. The only autopsy report on a congenital dislocation of the shoulder, found by Stimson, was of a double anterior dislocation reported by R. W. Smith.⁹ After reading Smith's paper, published in 1847, I would agree with Stimson that in all probability this was not a case of congenital dislocation. In my opinion, the great majority of dislocations of the shoulder in adults are anterior because they are due to hyperabduction, which can produce only an anterior dislocation. The few posterior dislocations that do occur in adults, are probably due to direct violence pushing the humeral head backward. The fact that practically all dislocations of the shoulder occurring at birth are posterior, is probably to be accounted for by the same mechanism, as the child is coming through the birth canal. Lange, in discussing Finck's idea of a preglenoid dislocation in these cases, maintained that a dislocation of the shoulder, to occur at all, must be complete, and that such a dislocation in the new-born could be shown by the X-ray. Neither Finck, nor any other writer, he says, has established in this way, a dislocation of the shoulder in the new-born. He adds that the application of strong force during birth would result rather in an epiphyseal separation than in a dislocation. There would be much force in these statements, if we ignored the fact that all dislocations are not produced in the same way, and this fact has not received much attention. The anterior dislocations are the result of indirect violence exerted through hyperabduction of the humerus and the first strain at the shoulder comes on the axillary portion of the capsule which must tear before a dislocation can take place. I have tried in several infant cadavers to produce an anterior dislocation by hyperabduction and have obtained each time an epiphyseal separation or a fracture of the upper end

⁹ Fractures and Dislocations, 1847.

of the humerus. The capsule was stronger than the humerus as suggested by Lange. Direct pressure backward on the head is not likely to fracture it. The X-ray will not show a typical dislocation of the shoulder in these subluxations, because they are not typical as we know dislocations (see Figs. 5, 6 and 7). The X-ray will, however, show a slight downward displacement, and a change in the normal shape of the head, probably due to abnormal pressure and retarded growth. I agree with Lange, for the ordinary dislocations in adults, that they must be complete. The humeral head cannot remain resting on the glenoid margin in the subluxated position. It must glide over into the completely dislocated position or back to its normal place in the socket. But in these posterior dislocations associated with obstetrical palsies the head remains resting on the posterior edge of the glenoid cavity in the subluxated position because of the obstructing anterior portion of the acromion bent down by the same force which pushed the head backward at birth, and possibly by a coracoid process that was also pressed downward and backward. The head cannot jump back into the socket because of this obstacle. Here is a very satisfactory explanation for some of the obstinate difficulty in reduction, encountered by Whitman and Fairbank, and which I found in four of my cases. Other obstacles develop in time, as from contracted muscles and other soft tissues and changes in the head of the humerus and glenoid cavity. In the early stages this abnormal portion of the acromion may be the only serious obstacle to reduction. In one of my cases, a boy ten months of age, I removed this obstruction and then had little difficulty in accomplishing the reduction or in maintaining it. In two other cases complete reduction could not be accomplished even after removal of this obstacle. Since this abnormal condition of the acromion was present in all nine of my obstetrical palsies with posterior subluxation, I have no doubt that it will be found by others.

In looking for the subluxation, the best sign, is the absence of the normal prominence of the upper end of the humerus, in front of the acromion, as determined by the palpating finger,

together with the presence of an abnormal prominence just below and behind the posterior margin of the acromion. The degree of displacement varies considerably in my cases, although in none is the dislocation complete. The amount of fat and the mild grade of displacement, may make the conformation of the shoulder so nearly normal that the subluxation will be overlooked unless one has in mind the possibility of its presence and makes a careful examination. I overlooked it in two of my early cases.

If a dislocation of the shoulder in the adult produces a brachial paralysis without rupture of nerve fibres, a dislocation in the new-born is much more likely to do so because of the much more delicate and sensitive muscles and nerves. This will account, I believe, for an interesting difference between the two groups of cases. In the new-born the paralysis seems to be complete in most or all cases for a time. In the adults, it is rarely complete. In both there is usually a tendency to rapid improvement. In a case, without dislocation, seen with Dr. L. C. Peter, about six weeks after birth, the palsy had almost entirely disappeared, yet immediately after birth the palsy was complete, according to the statement of Dr. Peter, who is a neurologist of experience. It is generally agreed that in the majority of cases, the paralysis spontaneously recovers. Reports of cases in which the extent of the paralysis is made evident by electrical examination, are conspicuous by their infrequency, and in this respect they are very similar to the adult brachial palsies from injury to the shoulder region. Fairbank found that in most cases the paralysis had largely disappeared by the end of the second month, as determined by electricity. In his series of 40 cases, the nerves appeared to have completely recovered or showed every sign of recovering at the time of his report in, at least, 60 per cent. of the cases. In several of the remaining 40 per cent., the residual paralysis affected one or more muscles of the forearm only, the bulk of the paralysis having entirely disappeared. It should be borne in mind that 14 of his 28 subluxation cases were in the first year of life, how many of the other 12 cases without subluxation he does not say. The

inference is that more than 60 per cent. of all his cases recovered from the paralysis completely, at a later period. Sherren says that about 70 per cent. of his cases recovered spontaneously, and that in many the paralysis had completely recovered at the age of three months, before which period he did not test the electrical reactions. But spontaneous recovery from the paralysis does not mean a functional recovery, as in 28 out of 37 of Fairbank's cases, there still remained a subluxation, which is not mentioned by Sherren. I have seen two cases in which the relations of the skeleton were normal at the shoulder and throughout the whole limb. In one case as already stated the child had recovered almost full use of the limb in about six weeks. I have seen this case recently and normal function and development have been present so long that the mother has almost forgotten about the palsy. In another case, now an adult, the patient has had full function in the previously paralyzed limb for many years. She has a clear recollection of the palsy in her early years, but it disappeared so gradually that she does not know now how long it lasted. This suggests what I believe to be true, *i.e.*, if the shoulder-joint relations are normal in a case of obstetrical palsy of the usual type, the limb will gradually recover full motion and function and will have its full growth. In a third case, the sister of one of my patients with subluxation, the shoulder-joint relations are normal, but there is some limitation of extension at the elbow indicating that an injury occurred there at birth. Now the function of the limb is practically normal, and such impairment as still exists is to be accounted for by the limitation of movement at the elbow.

If in an adult the dislocation remains unreduced, the associated palsy gradually disappears, but never completely. After years of persistent effort the power and motion may recover to a remarkable degree. The improvement in motion is responsible for the improvement in power, and the return of normal power is impossible because the return of normal motion is impossible. Stimson gave as one of his reasons for thinking that the most frequent variety of congenital dislocation of the

shoulder was probably traumatic (and not secondary to paralysis), "that the limitations of motion closely resemble those of the similar traumatic dislocations in adults." As I see these cases of obstetrical palsy with posterior subluxation at eight to ten years of age, especially the two which were incompletely reduced about two years ago, they are nothing more than old unreduced dislocations of the shoulder. Such limitation of movement and power as is present, is to be explained by the displacement in the shoulder-joint. Both Whitman and Fairbank believe that sometimes the displacement in the joint is the only obstacle to complete recovery. I believe that this is true of practically all early cases. In long standing cases, permanent changes have taken place, particularly, in the bones, and in some cases there is a marked lack of growth in the limb. Reduction of the dislocation will improve these very much, but of course will not restore the limb to the normal. It cannot be expected to restore the normal shape of the humeral head and glenoid cavity, although these may improve from long continued movement with the bones in normal contact with each other. While the affected limb may be shorter than the other and atrophied, we may expect improvement in size and strength. This has resulted already in my second case operated on, to a very satisfactory degree, and in the first case, the improvement is almost as satisfactory (see Figs. 8 and 9). It is of interest that the displacement in the first is more marked than in the second.

In one of my cases there was the usual posterior subluxation of the shoulder with the bent down condition of the acromion, and in addition an anterior dislocation of the head of the radius (see Fig. 10). Fairbank had a similar case and thought the radial dislocation congenital in origin. I prefer to believe that, like the shoulder dislocation, it occurred at birth and is traumatic in origin. Electrical examination by Dr. J. W. McConnell, did not reveal that any muscle was completely paralyzed, but while the muscles of the arm from the shoulder to the elbow showed the same atrophy and weakness as in the other cases of subluxation of the shoulder at about the same

age, unlike these, from the elbow to and including the hand, the atrophy and weakness were much more marked than above the elbow. This suggests that the muscles from the shoulder to the elbow suffered the usual palsy from a subluxation of the shoulder and from the elbow down they suffered the combined palsy consequent upon the shoulder and elbow dislocations. In Case IV, in addition to a subluxation at the shoulder, there is an abnormal prominence about the radial head and a turning of the forearm inward in the cubitus varus position. In Case VI, there is a considerable limitation of extension at the elbow (see Fig. 11). It becomes more than probable, therefore, that the elbow as well as the shoulder suffers from trauma in some births. In Case III, the hand is fixed in flexion, which I am inclined to ascribe to contraction of the flexors, although I have not had the opportunity of studying this case carefully.

In this discussion, I have not considered two groups of cases, those in which the brachial plexus is injured without flail-joint and those with flail shoulder-joint. I excluded the first because none of my cases seemed to be in that class, and the second or flail-joint type, for the same reason and because they seem to be exceedingly rare. Whitman mentioned the latter, but not in such a way as to infer that any of his cases were of this kind, and Fairbank did not mention them. I have not seen any, nor have I seen a report of such a case in the literature, although my search was not at all thorough. If in a case of obstetrical palsy, I found the anatomical relations at the shoulder normal, no other deformity in the bones of the limb, the sensation normal, and after several months the electrical reactions normal, I would not worry much about the ultimate outcome of that case. Any limitation of movement in the shoulder-joint from contraction of the capsule would gradually disappear, I believe, from persistent manipulations by the mother or nurse, and the normal function of the whole limb would recover, in time, completely or practically so. From my experience and study of obstetrical palsy, it seems to me that we have been paying too much attention to the brachial plexus and too little to the skeleton. Obvious injuries of the

skeleton, like fractures of the clavicle and humerus, both of which were present in one of my cases, receive attention, but the less obvious injuries, especially those of the shoulder-joint, have been largely overlooked. In any case I would look first to the shoulder. If this were the general teaching and practice, I believe that there would be much fewer permanent obstetrical palsies. If there was present a subluxation of the shoulder and the physician in attendance at birth had this possibility in mind, he would probably find it in most cases and would probably reduce it immediately. The palsy would then gradually disappear, although this might require months or years. If the dislocation is first recognized months or years after birth, the first consideration should be to reduce it. But while the condition has received little attention as yet, the work of Whitman and Fairbank show that the subluxation is very difficult of reduction. Of his 28 cases, Fairbank could reduce only five without operation. Whitman, who used the non-operative method of reduction, found that in the more extreme cases it is impracticable to complete the reduction at one sitting. He applied a plaster case after the first attempt and undertook the further correction after an interval of two weeks. In all the cases, he says, there is a strong tendency to return in some degree to the original posture. I have had, as yet, the opportunity of attempting reduction in only five of my nine cases with subluxation. In my first and second I failed to reduce completely by operation. In the third, I tried the Whitman non-operative method of reduction after exposing and removing the obstructing portion of the acromion, and accomplished complete reduction rather easily. But soon after the removal of the case the subluxation recurred and the limb took the position of internal rotation again. It could easily be reduced again by rotating the arm externally. I attribute this result to the fact that the posterior part of the capsule was made longer than normal by the subluxation and was not shortened after the reduction. The abnormal changes in the humeral head and glenoid cavity from the ten months' duration of the dislocation, probably favored the gliding of the head into the dislo-

cated position. I expect later to shorten the posterior part of the capsule and the overlying rotator tendons in this case. (This has been done since the reading of the paper.) In my fourth attempt at reduction, I merely removed the obstructing portion of the acromion, without shortening the posterior part of the capsule and rotator tendons, because I was satisfied that this would not suffice to hold the head in its normal position in internal rotation which, in this case, forced the head into the subluxated position every time against any resistance I could safely offer. The humeral head was much altered in size and shape and I preferred to permit a greater range of movement at the shoulder, *i.e.*, of the head from the normal position to the subluxated, in the effort to obtain a wider range of movement of the arm in external rotation and abduction. The findings in the fifth case differed also from the others. As we are only now beginning to appreciate the frequency of these subluxations, the best method of treating them has not yet been worked out. It is likely that immediately after birth non-operative reduction will be easy, that the difficulty will increase as the child grows older and the prospects of complete recovery grow less. My first two cases, which I have been able to watch for two years or more, and particularly Case IX, show that after years much improvement in motion and power can be obtained. It is of much importance that these young patients can afford to wait a long time for the gradual return of power.

Lange in 8 cases, following Hoffa, improved the usefulness of the arm, especially external rotation, by doing an osteotomy below the middle of the shaft of the humerus and obtaining union with the lower fragment in external rotation. The object was to overcome the obstinate internal rotation. The operation was followed by much improvement; for instance children, who could carry the arm only to the waistline before operation, learned to comb the hair, carry the hand to the mouth, button the clothes at the back, etc. But my patients, even after incomplete reduction of the subluxation by operation, showed much more improvement than this, they could

carry the hand higher and had nearly full external rotation. Case IX with a mild subluxation has recovered almost a normal arm from exercises alone, without reduction of the subluxation. Osteotomy does not improve motion of the arm, which can only be done at the joint, but merely improves external rotation at the expense of internal rotation. It should be borne in mind that Lange attributes the limitation of movement at the shoulder, to an epiphyseal separation of the upper end of the humerus, in these obstinate cases. I have no doubt that the existence of subluxation in these cases, will become generally recognized, when it will become obvious that the best place to improve the movement of the arm, is at the shoulder-joint and not at the middle of the humerus.

Conclusions.—In the great majority of cases of obstetrical palsy of the upper extremity, the primary cause is not rupture of the brachial plexus but an injury to the shoulder-joint, the plexus and its branches becoming involved in the adjacent axillary inflammation. Electrical examinations will fail in most cases to demonstrate actual nerve paralysis after two or three months and before that time they are not advisable (Fairbank and Sherren). Detailed reports showing accurately the extent of the nerve paralysis by electrical examination, are conspicuous by their infrequency.

In most of the cases in which the brachial plexus has been exposed by operation above the clavicle, rupture of the plexus has been assumed because of the presence of adhesions about the plexus and thickening of the cords. The very few cases in which the roots have been found ruptured, need further confirmation because of their very small number and because of the great difficulty in dissecting accurately the delicate and interweaving roots. Fairbank, evidently, experienced this difficulty and Boyer, in an autopsy, found the plexus impossible of good dissection because of the abundance of tough adhesions. The presence of these adhesions is best explained, in my opinion, by the extension a few inches upward of the

blood, synovial fluid and inflammatory exudate, from an injured shoulder-joint. Lange by operation in the axilla, found the cause of the paralysis to be the embedding of the branches of the plexus in thick connective tissue in the axilla, and found also a diminution and deformation of the head of the humerus.

The best evidence showing the primary cause of these obstetrical palsies is only now becoming properly recognized, *i.e.*, the frequent association with an obstetrical palsy of a posterior subluxation of the shoulder-joint. The presence of the bent-down condition of the acromion cannot be explained on the basis of a brachial plexus injury and, in my opinion, will prove to be the key to the whole situation (change in the shape of the coracoid process may also result from the pressure). Its presence in all of my cases with posterior subluxation, makes it practically certain that it and the subluxation are due to direct pressure by the maternal pelvis during birth and that the joint injury is the primary cause of the palsy. The most striking evidence of a shoulder-joint origin of the palsy in my cases is afforded by the progress of the condition in them after the improvement of the joint condition. The nine cases in which a more or less severe palsy was permanent, showed a posterior subluxation of the shoulder-joint in each. In two of the three cases in which there was no subluxation, full function has returned. In the remaining case, there is full power of all the muscles and full motion of all the joints, except the elbow, the only impairment of function being due to the limitation of extension at the elbow.

Immediately after birth the reduction of the subluxation will probably be easy. After a few months it becomes very difficult, probably because of the obstruction offered by the bent-down portion of the acromion, possibly change in the coracoid process, and by the changes in the surrounding soft tissues. When the abnormal portion of the acromion becomes ossified, it should prove to be a practically insuperable obstacle. The first indication at any stage is to reduce, the next to obtain the best possible motion at the shoulder-joint.

Obstetrical palsy without dislocation, in most cases, will be associated early with limitation of abduction and external rotation which will gradually disappear, the rapidity depending upon the force applied in stretching the contracted capsule and other soft tissues. The palsy will also gradually disappear but will continue for some time after the motion is complete. If an existing subluxation is reduced immediately after birth, complete recovery will probably follow in the same way. If reduced later, complete recovery will be prevented according to the degree of permanent change in the bones and other tissues from the continuance of the subluxation. The condition which develops is very similar to that associated with an old unreduced dislocation of the shoulder in an adult, the lack of growth being due chiefly to the interference with function during the growing period.

The chief responsibility in these cases, according to my view, will fall upon the physician in attendance at birth. The failure to recognize the frequent occurrence of these subluxations is due to their peculiar obscurity, but when once suspected they can be detected by careful examination. The recognition of the absence of the humeral head or tuberosities immediately in front of the acromion and of the presence of an abnormal prominence behind the acromion, is sufficient for diagnosis. The association of anterior dislocation of the head of the radius, abnormal prominence of the radial head, limitation of movement in the elbow, as found among my cases, indicate that the elbow is also subject to injury at birth. Injuries of the skeleton of the upper extremity, associated with obstetrical palsy, offer a fruitful field for further study. I fully agree with Lange when he says that "the day for the let alone treatment of obstetrical palsy has passed by."

In conclusion I wish to express my indebtedness to Professor G. G. Davis for his encouragement shown in the transferring to me of three cases with posterior subluxation from his service in the University Hospital, and to Dr. J. W. McConnell for his interest and assistance, as a neurologist, in my cases and in other phases of the work.

CASES OF OBSTETRICAL PALSY WITH POSTERIOR SUBLUXATION OF THE SHOULDER-JOINT.

CASE I.—Reported in ANNALS OF SURGERY, January, 1911.

CASE II.—Girl, ten years of age, referred by Dr. A. G. Tinney. Has been reared by grandparents and nothing is known of the birth except that the right arm has been palsied since. It is very little shorter than the left, but is much atrophied, held in marked internal rotation, and can be rotated externally only to a slight degree. Can carry the limb at the side of the body, but when unconscious of being observed, as at play, the forearm is flexed in front of the body, the arm is held in slight abduction and the shoulder is depressed, which position attracts attention to the crippled condition. Cannot flex elbow to a right angle, can abduct arm at shoulder to about a right angle but passive abduction cannot be carried much further. Seems to have some power in all the muscles of the limb, but all are weak, some weaker than others, dorsal flexion of the wrist being particularly weak. She generally uses her left hand because of the difficulty in using the right. For instance, she cannot raise her hand high enough to write on the black-board at school, except with much straining, and when she does the whole limb trembles and her writing becomes illegible. The anterior and outer portion of the acromion is bent downward and seems to be almost directly in contact with the humerus, but this was not observed until after the operation.

Operation (August 8, 1911).—At University Hospital, in service of Professor Edward Martin. With arm in abduction, capsule exposed by axillary incision between coracobrachialis muscle and large vessels and nerves, and subscapularis muscle divided. Capsule opened anteriorly from upper to lower part of joint. Finger in joint felt nothing abnormal except poorly developed and irregular head. Anterior and upper portion of glenoid margin removed with gouge and scalpel, to permit the head to be pushed upward and forward to its normal place, but this met with only partial success. Almost full external rotation was obtained. Patient then turned over and posterior portion of capsule exposed by incision along posterior margin of deltoid and division of tendons of infraspinatus and teres minor. Capsule divided, head pushed upward and forward, and an effort made

to hold it there by shortening of the capsule and divided tendons. Anterior part of capsule and subscapularis muscles sutured, and then both skin wounds, a small drain being left in each. Dressings. Arm fixed in full abduction and external rotation by a plaster case. Both drainage tubes removed in 48 hours, and primary healing obtained. Case removed August 20 and arm brought to side of body, gradually by changing the angle of fixation. When all fixation was removed, there was a severe palsy of the whole limb, but sensation was good throughout. Could flex and extend little finger and thumb, but could not move other fingers. Could not flex or extend elbow or rotate forearm. This palsy gradually disappeared and in about six weeks the power was better than before the operation. At the present time it is almost as good as in the left, and she has schooled herself to use it in preference to the left, although there is still considerable mechanical obstruction to movement at the shoulder, probably due chiefly to the bent-down condition of the acromion against which the humerus impinges, and to the incomplete reduction of the dislocation. Whereas before operation her crippled condition was apparent, practically all the time, it is evident now only when she raises both arms up above her head.

CASE III.—Boy, nine years of age. Referred by Dr. H. D. Beyea. Birth difficult, lasting about 60 hours, instruments finally being employed. The physician who delivered the mother, told her that the shoulders were very broad and that, in using instruments, he had pulled so long and hard that he was exhausted. Paralysis of right arm noticed by nurse during first bath. The only deformity noticed at the time, by the physician, was a turning inward of the left foot, which soon disappeared without treatment. As the child grew the whole right upper extremity gradually became shorter than the left, but developed some power in various parts. It is now atrophied as a whole, but much less above the elbow than below. There is a posterior subluxation of the shoulder, and a complete dislocation, anteriorly, of the head of the radius (See Fig. 10). This dislocation can be reduced almost completely, but recurs quickly when the pressure is removed. The anterior portion of the acromion is bent downward. There is a very marked compensatory hypertrophy of the whole left upper extremity.

At the University Hospital, January 22, 1912, an operation

was done to prevent recurrence of the radial dislocation. The radial head was nearly normal in shape as was the capitellum of the humerus. The lesser sigmoid cavity of the ulna had lost its normal concavity and was somewhat convex, and there was an abnormal prominence on the ulna just below. This was removed, the lesser sigmoid fossa reshaped with a chisel and curette, and the soft tissues cut away to permit the radial head to occupy its normal position. It was held in place by overlapping of the divided orbicular ligament. Wound closed. Fixation of limb at a right angle by an anterior splint. Primary healing. Splint removed on nineteenth day. February 17, 1913, there was improvement in gripping power of hand, and slight rotation of forearm, which parents had never noticed before operation. When I saw the patient again a few months later, the dislocation of the radius had recurred and all improvement in power of limb had stopped. My object had been to prevent recurrence of the elbow dislocation with the hope that it would be followed by a sufficient return of power in a few months to warrant the attempt at correction of the shoulder dislocation. The failure with the elbow condition caused the parents to decline further interference. If I had had a second chance to operate, I would have excised the head of the radius, and if permitted, would have attacked the shoulder later.

CASE IV.—Boy, four months and three weeks old. Obstetrical palsy of right arm, with posterior subluxation of shoulder and turning down of anterior and outer portion of the acromion. Mother very obese. Has had four children and all were delivered with instruments, but the birth of this child was the most difficult of all. After the head was born there was considerable difficulty in delivering the rest of the body, particularly, the right or now palsied arm. Immediately after birth the child was much cyanosed and recovered its normal color only after some effort and time. The paralysis of the right arm was observed soon after birth and was complete. There has been very little return of power since. For past five weeks, has been receiving electrical treatment in the nervous dispensary of the University Hospital, by Dr. J. W. McConnell, and seems to have improved faster in that time. The patient had been referred to the nervous dispensary from the orthopædic department, by Professor G. G. Davis, who later turned it over to me. The child could lift the

whole limb forward about 30 degrees. There was a well-marked wrist drop, but it was not noted at this time that there was any fixation of the wrist in flexion. There were no movements in the fingers except very slight when the palm was tickled. There was marked limitation of passive abduction and external rotation at the shoulder-joint. These notes were taken October 25, 1911, the first time I saw the child, but the parents did not return with it, and I did not see it again until August 18, 1913, when I looked it up to learn what its condition was. There had been a considerable improvement in power in the whole limb, although it was still a much crippled limb. Voluntary abduction at the shoulder to about a right angle, and passive to about 160 degrees. According to my recollection and the statements of the parents, this was a considerable improvement. The whole limb was still held in marked internal rotation and passive external rotation was very much limited. There is an abnormal prominence of the head of the radius and the forearm is turned inward in the cubitus varus position. There is some limitation of extension at the elbow. The hand occupies the wrist drop position and cannot be extended passively to the straight position, although there is considerable gripping power in the hand. The parents are not yet disposed to permit anything to be done to the limb.

CASE V.—Boy, ten months old, referred from orthopædic department to nervous dispensary of University Hospital, to be examined by Dr. McConnell, and then to me. Weight at birth said to be 13 pounds. Mother small, weight 108 pounds. Birth difficult. Head presentation. Instruments used. Right arm completely helpless immediately after birth. Now has characteristic internal rotation of whole limb, and passive abduction is much limited. Can hold light objects in hand. Has a little power in wrist and elbow, and raises limb at shoulder almost to horizontal. Atrophy of whole limb, but not extreme, and limb slightly shorter than its fellow, which seems to show slight compensatory hypertrophy. Normal angle on outer side at elbow is lost. Dr. McConnell observed a mild grade of posterior subluxation at the shoulder, which is evident only on careful examination. He also thought he detected a turning downward of the anterior and outer portion of the acromion, which I was satisfied I could feel, but the mild grade of dislocation and the amount of fat made the shoulder so nearly normal in conformation that it

would be necessary to have the possibility of the subluxation in mind and to examine carefully for it in order to find it.

The patient was etherized at the University Hospital, in the service of Professor Edward Martin, August 7, 1913. The subluxation was now more obvious and the bent-down condition of the acromion readily felt. By manipulating the lower end of the humerus outward in abduction and backward with the right hand, and using the thumb of the left hand as a fulcrum behind the humeral head, the latter was forced almost if not to its normal position where it could be felt in front of the acromion. It was evidently covered in great part by the abnormal portion of the acromion, which obscured it. When the pressure was removed, the subluxation quickly recurred. An incision was made about an inch and a half long, along the anterior portion of the acromion. This exposed a triangular projection of the acromion downward for about three-quarters of an inch, which was cartilaginous. The deltoid was detached from its margins, when it was easily pushed upward by the handle of the knife. The humeral head was then rather easily pushed forward and upward to its normal position, when there was a considerable gap between the cut margin of the deltoid and that of the abnormal portion of the acromion from which it was detached. As this was easily bent upward it was not removed except for about a half inch of its tip, and no attempt was made to close the gap between it and the detached deltoid. The skin wound was closed by catgut sutures, a dressing applied, and the limb fixed by a plaster case, with the arm at the side, the elbow in right angle flexion and a little posterior, and the humeral head pushed upward and forward to its normal position. The case was removed August 28, and the arm allowed to hang at the side. The humeral head was in good position. Six days later, the arm hung in the internal rotation position and the head was in the subluxated position, although by external rotation it easily took the normal position. It seems obvious that the posterior portion of the capsule must be shortened before the humeral head will be prevented from slipping back into subluxated position. I had hoped that this shortening would have resulted from the traumatic inflammation following the efforts at reduction and the rest in the fixed position. The change in the bones from the long continued pressure in the abnormal position probably had something to do with the tendency to recurrence of the

subluxation. (The posterior portion of the capsule and overlying rotators have been shortened since the printing of the paper.)

CASE VI.—Referred from orthopædic department to nervous dispensary of University Hospital and then to me. Boy, five years of age. Birth difficult. Instruments used. The attending physician told the mother that the arms were engaged over the child's head in what he called a "locked labor." A neurologist diagnosed a rupture of the brachial plexus and told the mother that it was the most complete he had ever seen. He exhibited the case before a society, when the child was a few weeks old. There is a posterior subluxation of the right shoulder and the anterior portion of the acromion is bent downward. There is marked limitation of external rotation and of abduction, the limb being held in internal rotation. It is considerably shorter than its fellow and there is some limitation of extension at the elbow (see Fig. 11). The hand is held in dorsal flexion and can be flexed, passively, only to the straight position. Has considerable grasping power in the hand and can raise the whole limb forward about 45 degrees at the shoulder. He seems to be unable to move the wrist or elbow, but in his efforts to do so he raises his whole limb from the shoulder and the wrist and elbow are held rigid. This rigidity and the contractions of the muscles of the arm and forearm which can be seen and felt show that he has considerable power which he cannot use.

Operation (at the University Hospital, September 4, 1913, in the service of Professor Edward Martin).—Semilunar incision along the margin of the acromion about $2\frac{1}{2}$ in. long. Anterior and outer portion of the acromion bent downward. It was cartilaginous and when the deltoid was detached from it, was bent upward easily to a level with the bony portion of the acromion. A good exposure was obtained of the upper end of the humerus through the wound. After repeated manipulations the head could be pushed upward and forward to its normal level with the arm in internal rotation, but could not be held there when the arm was turned in external rotation. There was strong resistance to external rotation until the subscapularis, some fibres of the coraco-humeral ligament and some of the lower fibres of the pectoralis major were divided, and considerable force was employed to overcome the remaining resistance. This resistance seemed to come chiefly from contracted soft tissues, including the

capsule and tendons, but the exact seat of the resistance could not be determined. Plain catgut sutures were passed through the skin and deltoid to close the wound. Dressing applied and arm fixed in abduction to a right angle and in full external rotation. Healing by first intention. Arm still in case.

CASE VII.—Boy, three years old. Difficult labor, but instruments not used. Complete palsy of right arm at birth. Much pain on moving arm from side in first few weeks. I saw this child about two years ago, going to its home for the purpose and finding the parents, who were foreigners, hostile to my desire for an examination. An operation on the brachial plexus had been advised soon after birth, and the power and movement of the limb had improved very much without it. My examination at that time did not develop a dislocation. Recently I traced the case to another part of the city, and found that the power of the limb was much improved. The mother states that it is not much below that of the opposite one. There was still, however, considerable limitation of abduction and external rotation in the affected shoulder, and on examination of it at this visit I discovered a mild grade of, but distinct, posterior subluxation with a bending downward of the anterior portion of the acromion. The improvement in power was so marked and so general that it seemed to me evident that the shoulder-joint condition was the only obstacle to complete recovery. My failure to recognize it in my first examination is easily explainable. It was only the second case of obstetrical palsy that I had seen and the subluxation was of much milder grade than in the first which I regarded as mild for a dislocation of the shoulder and which had never been recognized before, although the boy was then $7\frac{1}{2}$ years of age. It emphasizes the obscurity of the deformity and the necessity for care in the examination.

CASE VIII.—Boy, sixteen years of age. Difficult labor. Instruments used. Child was much cyanosed and recovered with difficulty. The physician in attendance said that the arm was broken, but he did not immobilize it. He advised rubbing with alcohol and said that the child would outgrow the palsy which affected the right arm from birth. At about eighteen months of age he was taken to a consultant who recognized a dislocation of the shoulder and said that the ligaments were twisted. Examination now shows that the muscles of the whole limb are very weak

and much atrophied, those of the shoulder and arm most, of the forearm less and of the hand least (see Fig. 15). Can raise the whole arm forward almost to a right angle. Flexion at the elbow is very weak, of extension much stronger but still much below normal. Rotation of forearm very weak. Movements of wrist and hand fairly strong but much weaker than of left side. The posterior subluxation is marked. The acromion has not the normal inclination from the anterior margin downward and backward but is more horizontal from before backward, indicating that it had sustained pressure from above downward, especially at its anterior portion which is normally the higher and would first receive the pressure from some object above it. At operation, the coracoid process showed distinct evidence of having been bent backward at birth, which may have protected the acromion from the marked bending downward seen in the other cases.

Operation (September 22, 1913).—At the University Hospital, in the service of Professor Edward Martin. Incision along anterior and outer margin of acromion, through the deltoid and exposing the upper end of the humerus. The sharp bending downward of the anterior portion of the acromion discovered in the two preceding operations, was not found, but the more horizontal position was evident. It seemed to be bent downward slightly as a whole. External rotation was stubbornly resisted until the subscapularis tendon and the underlying capsule were divided, when there was a considerable gap between the divided margins. With the arm in external rotation, the humeral head could be pushed forward so far that there was no prominence behind the acromion, but it could not be pushed forward and upward far enough to make a prominence in front of the acromion. It could be seen that this was prevented by the contact of the head against the coracoid process, which seemed to have bent backward considerably at birth. The patient was then turned over and an incision made through the posterior fibres of the deltoid, exposing the tendons of the infraspinatus and teres minor, which were divided near the greater tuberosity with the underlying capsule. The finger in the joint found the glenoid cavity flat from side to side but concave from above downward. With the head pushed as far forward as possible, the margins of the tendons and capsule were overlapped by catgut sutures about $1\frac{1}{2}$ inches. Both wounds closed with a small drain in the posterior

one. A dressing and a plaster case holding the arm in abduction to a right angle and external rotation, were applied. Case opened posteriorly and small drain removed on sixth day. No infection and dressings not removed since.¹⁰

CASE IX.—Girl, nearly five years of age. Very difficult labor. Instruments used. Shoulders very broad. After delivery of face, the severe cyanosis caused the physician to hurry the rest of the delivery by hooking his finger under the right axilla and pulling, when he felt and heard a "crack." Examination afterward excluded a fracture of the humerus which was suspected, but there was a complete paralysis of the whole limb. Dr. J. W. McConnell saw the patient at this time and has followed its course since. He diagnosed an injury of the brachial plexus, but asked me to see the case about two years ago. The power and movement had improved considerably by this time, but the whole limb was still very weak and there was considerable limitation of abduction and external rotation at the shoulder. Not finding a dislocation of the shoulder, I concluded that suitable exercises would restore normal motion and that this would be followed by normal power of the limb. These exercises have been kept up since. I had the second opportunity of examining this child very recently. I expected to find that normal motion had returned, but there was still slight restriction of abduction and external rotation, and this led me to examine the shoulder for an overlooked subluxation which I found together with a bending downward of the anterior portion of the acromion. It was of mild degree but distinct, notwithstanding which the persistent exercises had produced nearly normal motion at the shoulder and power of the whole limb. This case is a striking evidence of the influence of motion on the palsy. In size, shape and nutrition there is no discernible difference between the two limbs. It shows only in the mild restriction in the

¹⁰ The case was removed after six weeks and exercises began to restore motion. Whereas before operation the arm was so weak that the hand could not be brought to the mouth at meals, except by resting the forearm on the table and moving the head toward the hand; twelve days after removal of the case he wrote, "the stiffness is out of the arm so (far that) I can lift it halfway over my head without the assistance of the other. I can take off my hat, blacken my shoes and turn on the electric lights." I believe that this early improvement means, merely, that from the better joint conditions he can make more use of the power he had before operation.

range of movement in the affected limb. The subcutaneous fat is greater than usual for this age, and gives to the affected shoulder almost if not perfectly normal roundness. This is the second case in which I overlooked a mild subluxation on my first examination (see Case VII).

It may be argued that the failure to recognize the subluxation in this and Case VII in my first examinations shows secondary development of the subluxation from an injury to the brachial plexus. I am satisfied that it merely indicates a lack of familiarity with the condition at the time of my first examinations. Both cases were seen only a short time after I saw my first case, in which I recognized the dislocation only after a prolonged examination. I thought it of mild grade but, with the exception of Case VIII, it was the most aggravated of all my cases. The two that were overlooked are about the mildest. I saw Case VII at about 8 months of age, this one (IX) at about three years of age. Fairbank says that at the end of two months the paralysis has so largely recovered that electrical examination is usually unnecessary. Such a paralysis is hardly likely to develop secondarily a subluxation after 8 months or three years. My failure to recognize an existing subluxation in these two cases is easily accounted for by its mild grade, the amount of fat and my inexperience. I had not then developed what I regard as the pathognomonic sign, the absence of the normal prominence of the upper end of the humerus immediately in front of the acromion. This last case is a striking example of what can be accomplished in birth palsy by restoring motion to the shoulder-joint. Reduction of the subluxation is the great indication because perfect motion can occur only in a perfect joint.

THE FREQUENCY AND SIGNIFICANCE OF INJURIES TO THE ACROMION PROCESS.*

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RECENT studies of the etiology and pathological anatomy of certain injuries to the shoulder-joint have done much to make clear the causes of severe symptoms often found when gross lesions of the tissues are not demonstrable by examination. Such facts, however important, should not make us lose sight of that great class of cases in which some lesion to the bony structures of and about the joint may be shown, by X-ray if in no other manner.

The work of Ross and Stewart has called attention to the importance of sprain fractures in the causation of severe symptoms and has made it plain that the extent of a bony lesion by no means determines its immediate symptoms or sequelæ.

In acromial injuries we find a group of conditions often apparently trifling which, nevertheless, are of importance both as to the symptomatology of the lesion itself and because of the significance which a lesion may have in indicating the occurrence of other injuries.

Fracture of the acromion is a very common injury. An examination of the records of the German Hospital for eight years, from 1905 to 1912 inclusive, resulted in finding 89 cases of acromial fracture.

When there is a fracture of the acromion it is one of three classes: (1) A well-marked fracture of a considerable portion of the process; (2) a separation at the epiphyseal line; (3) a sprain fracture.

Of the cases mentioned it was impossible to determine

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definitely to which class the fractures belonged, except in those occurring during 1911 and 1912. Of the 40 cases demonstrated by X-ray in 1911 and 1912, 8 were fractures of a considerable portion of the process, 1 was an epiphyseal separation or separation at the epiphyseal line, 25 were sprain fractures, 6 could not be traced.

It is at once evident that fractures including a considerable portion of the process are few compared with the sprain fractures.

It must be apparent also that such fractures present no features as to diagnosis, etc., in any way differing from fractures in general. A typical example is shown in Fig. 1.

The separations at the epiphyseal line are, as far as causation and symptomatology go, merely a subdivision of the fractures of the first class (see Fig. 2).

The sprain fractures furnish the most numerous and in many ways the most interesting subdivision. True sprain fractures, by tearing due to ligamentous pull, are found in three locations: (1) most often at or above the acromio-clavicular junction; (2) at the insertion of the coraco-acromial ligament; (3) the upper surface of the acromion—usually the location of the smallest of the sprain fractures.

These sprain fractures have been noted in the order of frequency of their occurrence. Some of them are quite easily evident on the X-ray plates; others again are most minute. In several instances in which a sprain fracture was noted the diagnosis according to the X-ray plate seemed to me to be doubtful indeed. But a very minute sprain fracture cannot be demonstrated by X-ray, certainly not when only a few hardly perceptible fragments of the bone are pulled loose. It is beyond question that in the majority of instances the diagnosis was based upon substantial grounds.

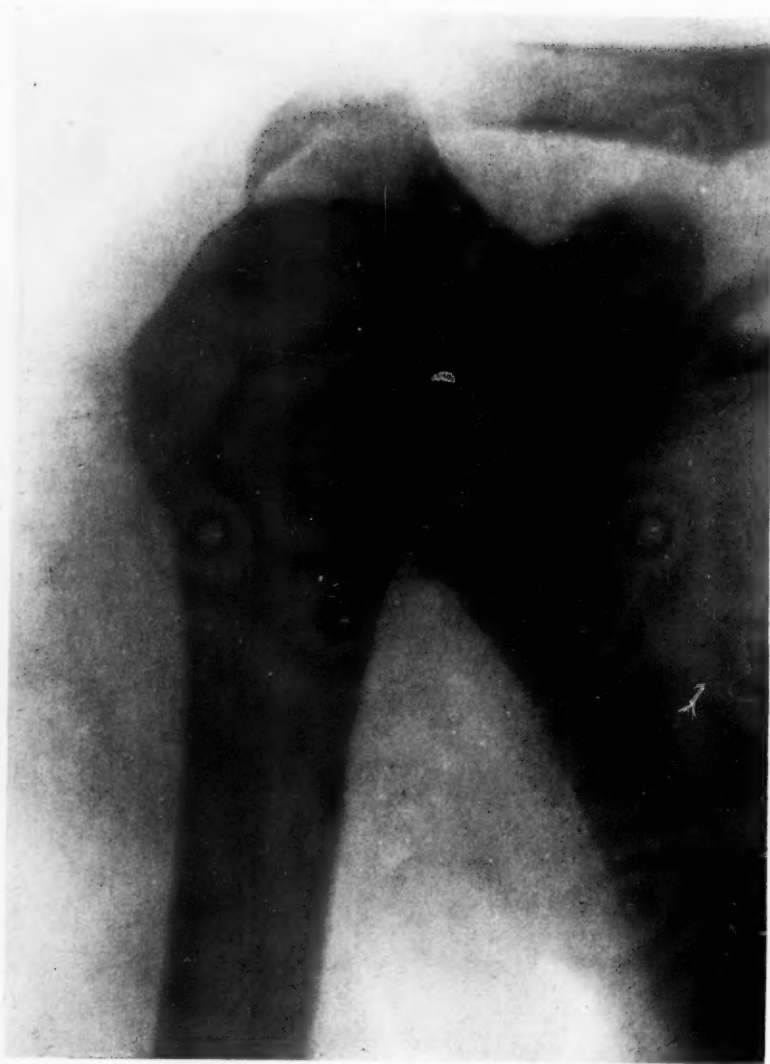
The fourth variety of sprain fracture or at least fracture of a very small portion of the acromial tip are due very evidently to a force exerted directly either (*a*) by the pressure of the humerus from below, or (*b*) by direct violence to the acromion process.

FIG. 1.



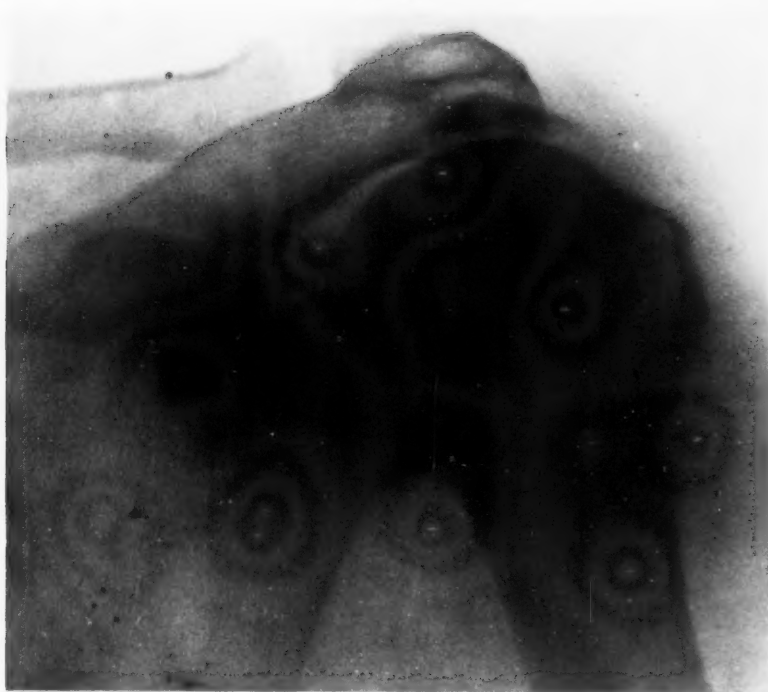
Fracture involving whole acromion process.

FIG. 2.

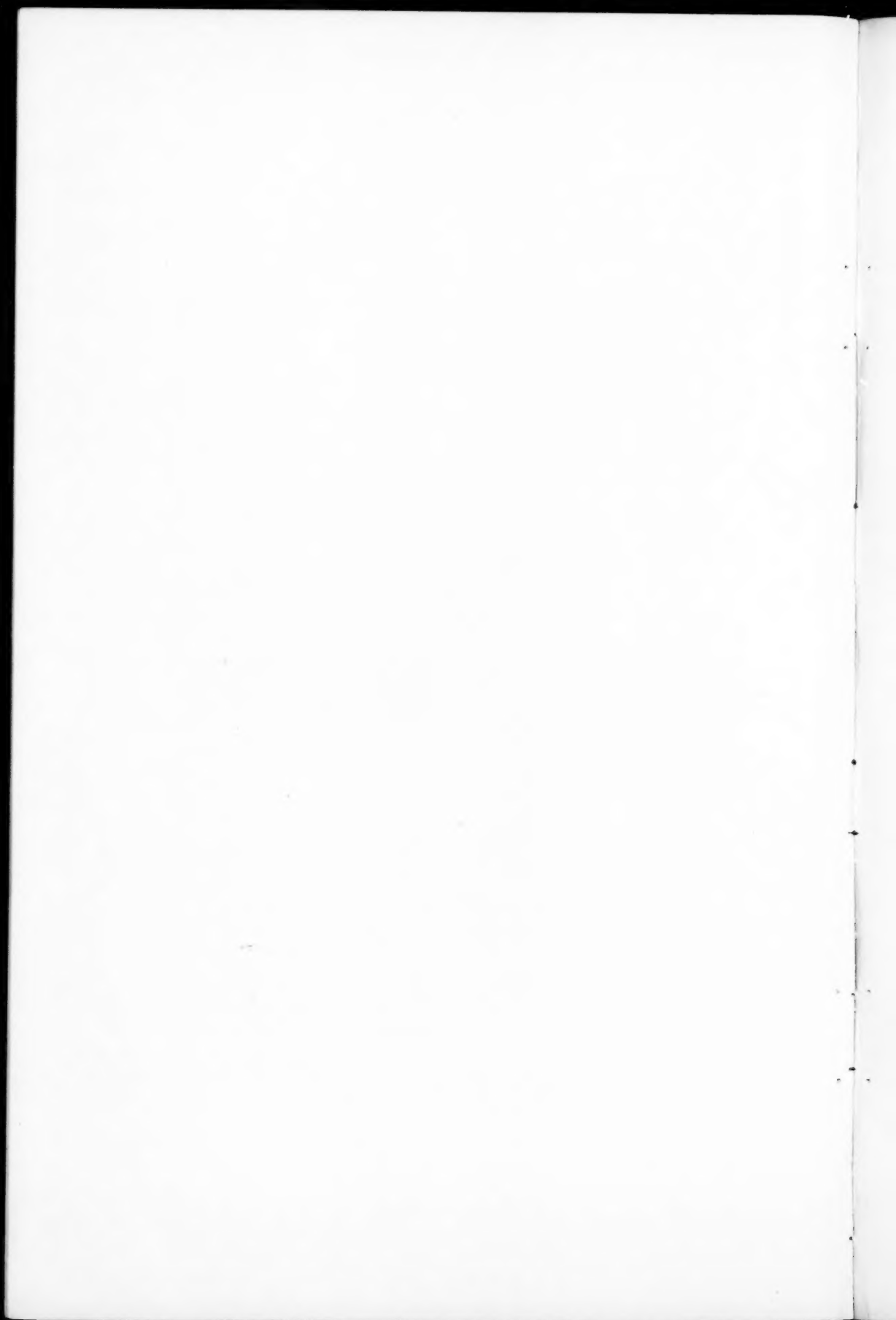


Injury to epiphyseal line of acromion with sprain fracture of upper surface of acromion.

FIG. 3.



"Chipping" of outer and lower portion of acromion process by force exerted through the humerus.



Some of them in extent and appearance are such that I find them in my records noted as "bruises of the tip of the acromion."

Of the total of 89 cases of fracture of the acromion, 18 were found with other lesions also demonstrable by X-ray. These were 3 instances of associated injury to the clavicle at its acromial end, 3 instances of fracture of the acromion with luxation of the acromial end of the clavicle, 3 instances of old luxation of the humerus, 2 instances of subluxation of the humerus, 1 instance of fracture of the greater tuberosity of the humerus, 1 instance of luxation of the head of the humerus and of the clavicle, 1 instance of luxation of the humerus with fracture of the clavicle, 1 instance of fracture of the head of the humerus with luxation of the clavicle, 1 instance of fracture of the "upper end" of the humerus, 1 instance of comminuted fracture of the surgical neck of the humerus, 1 instance of fracture of the coracoid process and of the head of the radius.

It will be seen at once that these injuries associated with acromial fractures, sprain fractures or otherwise, group themselves into two great classes.

1. Conditions affecting the acromio-clavicular junction.
2. Associated injuries indicating a violent trauma involving the upper end of the humerus and producing either a luxation of the humerus or a fracture.

Since sprain fractures of the acromion are by far more common than any other form of fracture of this part, and since most of the sprain fractures involve the acromion at the acromio-clavicular junction, it is not surprising that at times there should be a similar lesion of the acromial end of the clavicle. And, luxation so-called of the acromio-clavicular articulation, as shown by the X-ray, is but one step further in an acromio-clavicular disjunction. I have never seen such an occurrence in which this separation, accompanying merely a sprain fracture of the acromion, was clinically demonstrable.

Those cases of injury to the acromion in which we have, as in the old or unreduced luxations of the head of the humerus or the fractures of the humerus, evidence of great force ex-

erted upon the upper extremity of the humerus are of great interest because they furnish us probably with an explanation of the severe symptoms often accompanying what seem to be very insignificant acromial injuries.

Before considering these conditions we must take into account the method of causation of acromial injuries.

In all but a few instances in which there is some history of the injury, to be correct, in 8 of the total number of 89, a fall is given as the cause of the acromial lesion. In a considerable proportion of the cases the history stated that a "fall on the shoulder" took place. I believe that these histories are generally incorrect. Codman, who is in this supported by Thomas, has drawn attention to the unreliability of such statements as to shoulder injuries. It is no easy matter to fall in such a way that the first impact is upon the tip of the shoulder.

The only other explanation possible is that, where the acromial injury is not a direct one, it is caused by a force transmitted or applied by the humerus. The possibility of this I have seen mentioned by Dr. G. G. Davis. It is the frequency of this sequence of events that I wish to emphasize. Now since in a fall the arm is practically always thrown away from the body—abducted—we find that the force is applied to the acromion by the greater tuberosity of the humerus, the shaft of the humerus acting as the long end of the lever. It may be possible for a direct upward push on the humerus to do the same thing, the scapula being fixed. One of the series of cases I studied sustained the acromial injury while cranking an automobile and this may be such a case.

The force applied to the acromion may then (*a*) clip its outer end (as Fig. 3), (*b*) "spring" the acromio-clavicular junction, or (*c*) put too much strain upon the coraco-acromial ligament.

In any of these conditions, the acromial fracture or sprain fracture results.

It is evident, therefore, that a minor degree of acromial injury may be the net result in damage to the bony structure of considerable violence.

The association of acromial fracture with luxation of the head of the humerus gives rise to several questions of importance. I was very much interested in Dr. Thomas' explanation and demonstration of acromial injuries associated with the birth palsies of children, which he has found to be the result not of nerve lesions but of injury to the bony and ligamentous structures of the shoulder.

The fact that we occasionally find injuries to the acromion with luxations at the shoulder leads us to consider two possibilities: (1) That many cases of acromial injury are associated with luxations of the shoulder which become spontaneously reduced; (2) that the giving way of the acromion or of the structures attached to it accompanies trauma not quite sufficient to cause complete luxation, yet sufficient to injure the capsule of the joint and thus produce subsequent symptoms.

The spontaneous reduction of a shoulder luxation is beyond a doubt possible but in the majority of instances of acromial injury the examination fails to reveal evidences that actual luxation has occurred. We are thus thrown back upon the second possibility. It seems to me most likely that the application of a force in abducting the shoulder sufficient to produce a fracture of the acromion must be such as to produce an accompanying injury to the contiguous soft parts.

The symptomatology of acromial injuries, even of the sprain fractures apparently most insignificant, bears us out in this view.

A certain proportion of these cases show immediately after injury only the two symptoms referable directly to the acromial condition, *i.e.*, localized tenderness over the acromion at the seat of injury and pain on abduction. After the lapse of three or four weeks these cases recover, occasionally, however, requiring active massage and passive motion for an equal length of time before recovering full function. Most cases of such injury, however, run a more severe and protracted course. Tenderness over the acromion is persistent and pain here and throughout the shoulder is complained of. Abduction is limited and in at least two cases that have come to my notice but which

I was not able to examine, an apparently permanent disability of the shoulder resulted.

It must be evident that while a fracture or sprain fracture of the acromion should and does cause localized tenderness and pain, and pain upon abduction, these symptoms should cease with proper treatment, at a time when bony or fibrous union has taken place. The persistence of symptoms points to the existence of a concomitant shoulder condition. There has been much said concerning subacromial bursitis as causing stiff and painful shoulders. I have never seen any case in which I could make this diagnosis.

We must then consider the accompanying lesion as being, as has before been stated, a luxation of the head of the humerus, spontaneously reduced, or an injury just short of producing luxation but with similar injury to the joint capsule.

In the clinic at the German Hospital we have been fortunate in avoiding bad end results in cases showing fracture of the acromion, because we treat every case, however slight, of injury to the acromion by rest for three weeks, with early massage and later, if necessary, by vigorous active and passive motion.

In conclusion, then, I believe we may safely state:

1. That injuries to the acromion process are not infrequent.
2. That they are important not only because the injuries themselves cause more or less pain and discomfort, but especially because practically all acromial injuries are caused by indirect force applied by the humerus acting as a lever and therefore an injury of this kind to the acromion is evidence that there has been either luxation spontaneously reduced or, as is more often the case, a lesion nearly approaching luxation with a corresponding injury to the joint structures.

I am indebted to Drs. G. G. Ross and A. D. Whiting, my chiefs in the German Hospital Out-Patient Department, for permission to report these cases and to Dr. A. G. Miller, the radiographer of that institution, for his kindness in furnishing the plates I have brought and his many demonstrations on this subject to me, and to the Fellows of the Academy for the opportunity to present this paper.

STENOSIS OF THE PYLORUS IN INFANCY.

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THE subject of this paper will be considered in four divisions: First, a systematic statement of the facts of the disease; second, reasons why the treatment should be surgical; third, a consideration of two problems which have arisen in connection with the study of these cases; and fourth, a report of my own experience with this disease.

I. A SYSTEMATIC STATEMENT OF THE FACTS OF THE DISEASE.

The Pathology.—A pyloric tumor is always present. It is about the size of the terminal phalanx of a finger or thumb, oval in shape, smooth of surface, firm or hard, like cartilage. There are never adhesions about it. The lumen of the pylorus is narrowed. The longitudinal folds of mucous membrane are enormously hypertrophied, adding to the narrowness of the lumen. This tumor is caused by an overgrowth and an hypertrophy of the circular muscle fibres of the pylorus. The tumor is a muscle tumor; it represents an overgrowth of muscle tissue.

The tumor is as evident at autopsy as upon the living. It exists in the living, whether gastric peristalsis is present or not. It is no more evident to direct touch when gastric peristalsis is present than when there is no gastric peristalsis. It is a passive tumor. Muscle contraction is not necessary to its existence.

That the pyloric tumor encroaches upon the lumen of the pyloric canal is proven not only by the clinical signs in these cases but by direct examination of the stomach at autopsy and at the operating table. The tumor itself is sufficient cause for the obstruction to the pyloric canal. The obstruction is an an-

atomic one, and is not necessarily dependent upon physiologic causes. The significance of this fact will appear later.

All other pathologic changes are secondary to the obstruction caused by the tumor, viz., the thickened or stretched gastric wall, the dilated œsophagus, the empty intestine, the emaciated and wizened body of the baby.

The Etiology.—What is the cause of this tumor found at the pylorus in these new-born babies? This has been the subject of much speculation. The most likely hypothesis is, I think, the one that considers it a congenital anomaly. The tumor represents a congenital overgrowth of muscle tissue. In support of this view are the following considerations:

1. The earliest indications of the presence of a pylorus is in the third month of fetal life. There is, therefore, ample time for the growth of muscle tissue to take place.

2. There is one case recorded in literature by Dent of a pyloric tumor in a seven months old fœtus. The tumor shows the same structure that is found in the stenosis cases examined after birth.

3. The symptoms in these cases appear so near to birth that it is impossible to conceive of the overgrowth of muscle as having taken place between birth and the onset of symptoms. My youngest case was only 14 days old.¹ The tumor in this case was fully developed and as definite as those seen in cases 3 months old.

4. The tumor is associated occasionally with other congenital defects, such as imperforate anus and club foot.

5. Aberrant Brunner's glands that normally belong only in the duodenum have been found in the tumor at the pylorus. It seems to me therefore that the evidence at hand favors a prenatal or congenital overgrowth of muscle tissue as the best explanation for the tumor present in these cases of infantile pyloric stenosis.

Why talk of or consider the etiology? Because it is important to determine the significance of spasm of the pylorus

¹ Boston Med. and Surg. Jour., December 14, 1905.

which is said to occur in certain of these cases. If it is likely that a congenital overgrowth of muscle is the cause of the tumor, then spasm, which has never yet been known to have caused a hyperplasia, is removed still further from the field of symptomatology in these cases. I think it will appear as the facts concerning this disease are unfolded that spasm has but little to do with these cases of tumor obstruction.

The Symptoms.—The symptoms are those of obstruction. The patient is usually a healthy appearing breast fed boy. There is at first, often overlooked, loss of appetite. The baby does not care to nurse. Vomiting appears soon after birth or within the first two or three weeks. This vomiting is characterized by its persistence and its projectile character. It is the vomiting of obstruction. The quality of the food seems to make no difference with the vomiting, the vomiting depending rather upon the quantity taken. The amount of the vomitus depends largely upon the amount of the feeding. The material vomited is the food taken. The vomitus never contains bile, an excess of HCl, blood, mucus, or lactic acid. Because of the little material passing through the pylorus into the duodenum the baby is constipated. The dejections are consequently small in amount; there being very little milk residue the stool, consisting almost entirely of bile, pancreatic juice, and cast-off epithelium, is meconium-like.

There is a progressive loss of weight. The child has not been receiving sufficient nourishment to keep the weight up to the normal gain. Instead of the normal gain there is an actual loss. There may be erratic gains in weight which subsequently are lost. If the baby's abdomen is uncovered while the baby is feeding, or while the baby is taking water from the bottle, there will be noticed rather vigorous peristaltic waves passing across the upper half of the abdomen from left to right. This visible peristalsis is very marked in many cases. The stomach is contracting violently in the attempt to overcome the obstruction. If the abdomen is palpated from the side and from before backward, in about from 60 to 80 per cent. of the cases it will be possible to feel the tumor between the thumb and

finger. This will be noticed more readily just after the peristaltic wave passes the pyloric portion of the stomach. The tumor may be obscured by an enlarged liver and mistaken for enlarged lymphatic glands, or even for the lower pole of the right kidney. The stomach itself will be dilated, particularly if the baby has lived some time after the obstructive symptoms have been present.

The obstructive vomiting, the palpable tumor, the visible peristalsis, the meconium-like stool, the epigastric fulness, the continual loss of weight, these are the symptoms of pyloric obstruction in infancy. Despite experiments with feeding and the use of drugs of various sorts, the baby gradually wastes away and dies of starvation. The baby dies of pyloric obstruction.

This is the typical picture of an unrelieved pyloric stenosis in infancy, and it is the usual termination. The death certificates in cases of this kind in the past, and also to-day, are often signed by the attending physician, inanition, acute gastritis, infantile atrophy, gastro-intestinal catarrh, marasmus, dyspepsia or pyloric spasm.

Diagnosis.—The diagnosis in typical cases is comparatively easy. However, there are many cases of babies difficult to feed who may be suspected of having a pyloric tumor. Pediatricians have employed the term "spasm of the pylorus" in order to explain the obstructive symptoms seen in little babies who suffer from persistent vomiting, and in whom there is no demonstrable tumor. This idea of a spasm of the pylorus is a purely hypothetical notion introduced by clinicians to account for symptoms which they are otherwise unable to explain. There is little doubt that there is a group of cases difficult to feed which is fairly easily explained by the idea of pyloric spasm without the tumor. These supposedly pure spasm cases occur in bottle-fed, excitable, irritable, neurotic babies. The onset of symptoms is several weeks after birth. The stools contain fecal material. A pyloric tumor, if felt, is felt only when the gastric contraction occurs. The vomiting lacks the characteristics of the tumor cases. Cases of obstruction from pyloric spasm sometimes die from starvation.

The serious and desperate cases are the ones that may become confused with the true tumor cases. These desperate spasm cases may occasionally but very rarely require surgical treatment.

The employment of the X-ray for diagnosis in these doubtful cases is likely to prove of a good deal of assistance. The behavior of the stomach in a normal baby after the milk of bismuth has been introduced into it is definitely known. The behavior of the stomach in a case of pyloric obstruction due to tumor when the milk of bismuth is introduced is likewise known. If bismuth is introduced into the stomach of a baby having a supposed pyloric spasm, the behavior will be often different from the record in either of the other two conditions. This difference may be helpful in the differentiation of these conditions. Every suspicious case of pyloric obstruction in which there is doubt as to whether there is a tumor or not should be X-rayed. The subnitrate of bismuth may be administered by mouth and the stools watched for the appearance of bismuth crystals (Talbot). The appearance of these crystals in the stools will be indicative of something passing through the pylorus. If the stools are infrequent and small in amount, these facts in themselves may be significant.

The Prognosis.—The mortality of this disease is high. Most cases of congenital pyloric stenosis die of starvation. The question is how long will it take a small baby to starve to death while the family physician experiments with drugs and foods, which under the conditions are absolutely of no use. It will take about three months, and this is the usual length of life of these small babies. Of course, the degree of obstruction in these cases, as has already been pointed out, varies. A baby with considerable obstruction will live a shorter time than a baby who has less obstruction, other things being equal.

There are cases being reported each year of young adults who have suffered during infancy and childhood from partial pyloric obstruction. Such individuals reaching maturity after years of gastro-intestinal invalidism, poorly nourished, and probably under-developed,—such individuals are more fre-

quently than formerly being recognized as instances who as babies have had a partial pyloric obstruction and have survived despite the obstruction. Hezekiah Beardsley,² in 1788, reported the case of a child who had lived five years with a pyloric tumor, which was determined at the autopsy. Habersohn,³ Lebert,⁴ Landerer,⁵ Rudolph Maier,⁶ Dunne,⁷ Tilger,⁸ and Barling,⁹ all have reported cases of this sort.

II. TREATMENT.

I believe that the treatment of stenosis of the pylorus in infancy should be surgical as soon as the diagnosis is made, and for the following reasons:

That the pure pyloric spasm obstruction can be cured by medical treatment in a large proportion of cases is true. It is also true, so far as I am able to learn, that there is no case of true tumor which has yet been cured by medical treatment. So far as I am able to determine, no so-called medically "cured" case has even been proven to have had the disease, but on the other hand many cases of supposed "cure" have relapsed and have been subsequently treated surgically. The tumor has been demonstrated to exist and a cure by surgical means has followed. Those who advocate and practise the medical treatment of true tumor cases do so upon the erroneous hypothesis that muscle spasm is the chief cause of the obstruction. They lose sight of the fact that it is the tumor that obstructs. At best, medical treatment relieves only hypothetical spasm that perhaps accompanies certain tumor cases. Medical treatment does not effectively remove the primary cause of the obstruction.

The improvement in Heubner's series of cases and in the

² Beardsley Hezekiah: Trans. New Haven Co. Med. Soc., 1788.

³ Habersohn: Diseases of the Abdomen, 1862.

⁴ Lebert: Diss., Tübingen, 1878.

⁵ Landerer: Diss., Tübingen, 1879.

⁶ Maier, Rudolph: Virchow's Arch., Bd. cii, s. 413, 1885.

⁷ Dunne: Jahresbericht d. Jenner'schen Kinder-Hospitals, Bd. xix, 1881.

⁸ Virchow's Arch., Bd. cxxxii, s. 290, 1893.

⁹ Barling: The London Lancet, January 29, 1913, 1913.

cases of others who have thought that they have been treating tumor cases with success is to be accounted for upon the basis of mistaken diagnosis, or a temporary and not a permanent cure.

It was about 23 years ago that the pyloric tumor cases were first well described. During all these 23 years the physician has painstakingly striven to treat such cases by medicines and by carefully prescribed feeding. The estimated mortality from an expectant medical treatment is between 80 and 90 per cent. (Monier). It is upon this carefully studied medically-treated post-mortem material that much of our present pathologic knowledge of this disease is based. The medical treatment of the tumor cases has signally failed to effect a cure.

What has surgery already accomplished in the care of these cases? Surgery has gradually lowered the mortality in the treatment of these cases. The mortality, once high, very distinctly is decreasing. The first time surgeons attempted to treat this disease was in 1898. From 1898 to 1905 is a period of seven years. During this period gastric surgery was developing. Operative technic was unsettled. The choice of procedure adapted to certain conditions was undetermined. This was an experimental period for gastric surgery in the adult and absolutely a new field in infants. Several different operations were done by many operators for the same condition. The cases operated upon had gone almost the limit of life under medical experimentation. Is it any wonder that the mortality from surgical operation during this period upon such material was very considerable? The mortality for this period was 46.5 per cent. No apology is needed here for this mortality, for more than half the babies entrusted to the surgeon were saved by operation. Even this was a great improvement over the medical mortality.

Consider now the next 7 years, the period from 1905 to 1912. The lowering of the mortality under surgical treatment has been remarkable. I have not yet collected all operated cases during this period. I have three groups, however, which are fairly representative of the period.

1. The group of ten operated cases from the Pacific Coast, collected by Stillman. In this group six different surgeons operated. A posterior gastro-enterostomy was done in each case. Of the ten cases only one died.

2. The group operated upon by Richter, of Chicago. There were 9 cases, only one died.

3. My own group of 17 cases with 3 deaths, a mortality of 17.6 per cent. A total, therefore, of 36 cases with 5 deaths, or a mortality of 13.8 per cent.

The mortality of posterior gastro-enterostomy in congenital stenosis of the pylorus is low under the above conditions.

III. CONSIDERATION OF TWO PROBLEMS.

There are two important problems which this group of cases helps to solve.

(a) What is the effect of gastro-enterostomy upon the metabolism of the body? There are those who think that a gastro-enterostomy impairs digestion. The passage of the food through the artificial stoma is looked upon as a real harm to the individual. Digestion, they say, cannot proceed in the proper fashion and the individual will suffer because of such impairment of digestion.

In order to determine the effect of gastro-enterostomy upon digestion it occurred to me that these babies with congenital stenosis might serve for metabolism investigations. The work done by Dr. Talbot, of Boston, upon a series of babies who had had a posterior gastro-enterostomy done for a stenosis of the pylorus has demonstrated that in these cases there is no impairment of the digestion of fat, starch and protein. The details of these experiments together with the results, I have already reported with Dr. Talbot in a former paper.¹⁰ If to the chemical evidence thus obtained be added the clinical fact that all these babies, without exception, are apparently thriving and in perfect health, have lived several years following the operation and gained in weight and height, the evidence is overwhelming that in these human babies gastro-enterostomy

¹⁰ Surg., Gynec. and Obstet., September, 1910, pp. 275-287.

has no deleterious effect upon the metabolism as measured by the digestion of fat, protein and starch and the normal development.

These experiments serve to confirm the work of Cameron and Paterson and make it absolutely conclusive that in the otherwise normal individual a posterior gastro-enterostomy has no harmful effect upon digestion, so that the opposition to this operation cannot be based upon any such conception as stated above.

(b) The second question that arises in connection with these cases is what becomes of the muscle tumor at the pylorus; does it disappear as the child grows older? I think from the evidence at hand that it probably persists and does not disappear, and for the following reasons:

(1) Through the assistance of Dr. W. J. Dodd, of Boston, skiagrapher at the Massachusetts General Hospital, and instructor in Roentgenology in the Harvard Medical School, I have been able to obtain further X-rays upon this series of stenosis of the pylorus cases operated upon by me, and these X-rays show uniform findings. In every case, no matter how many years following operation, the bismuth meal is seen to pass through the stoma, and in only a very few is it seen to pass in slight amount through the pylorus. In other words, the obstruction at the pylorus, which has been proved in each of these cases to have existed, is demonstrated by the X-ray to still exist. The tumor is still present and still obstructs.

(2) It has been demonstrated by certain physiologists that if the pylorus remains open and is unobstructed the stomach contents will be forced through the pylorus even though an artificial stoma be present. On the other hand, it has been demonstrated that if the pylorus has been closed by some form of obstruction either partially or completely, the food will be forced through the artificial stoma in whole or in part. In these cases the food is seen to be going through the stoma and it is reasonable to suppose, therefore, that the obstruction still persists. In other words, the physiological evidence confirms the evidence from the X-ray.

(3) Bearing upon the persistence of the pyloric tumor after operation mention must be made here of the pathological evidence in the unique case of Morse-Murphy-Wolbach.¹¹ The facts are these: A boy baby with pyloric stenosis diagnosed by Dr. J. L. Morse, was operated upon by Dr. F. T. Murphy by a posterior gastro-enterostomy. The pyloric tumor was seen and palpated. The child lived 7½ months, weighing then 19 pounds. During this time the baby had developed as a normal child, was breast fed, had not vomited, and had had normal movements. When 8 months old the baby died of nothing connected with the operation. Fortunately a complete autopsy by Dr. Wolbach was secured. The artificial stoma was found patent and functionally efficient. The pyloric tumor—and this is the point of present especial interest—persisted and appeared as at the time of operation. Microscopical study found the tumor to be a true tumor of circular muscle fibre, hypertrophy. This is the only instance of congenital pyloric stenosis that has been studied so long as 6½ months following a successful gastro-enterostomy.

(4) Attention should be called to the increasing group of adult cases of partial obstruction reported by Beardsley, Landerer and Barling, and others, who have had symptoms of pyloric obstruction for many years and have reached young adult life with all the evidences of difficult feeding and impaired nutrition. These are cases with a pyloric tumor which has partially obstructed the lumen of the pylorus, not sufficiently to have caused death from starvation, but only sufficiently to have caused impaired digestion and malnutrition.

This evidence, then, from the X-ray, from physiology, from the post-mortem table, and from clinical observation, points pretty conclusively to the fact that the muscle tumor at the pylorus does not materially change. This is not a mere academic question, but it has, of course, a practical bearing, and places the surgical treatment of this condition upon a very firm basis.

¹¹ Boston Med. and Surg. Jour., 1908, clviii, p. 480.

IV. REPORT OF CASES.

I wish to report here in detail the 17 cases treated by me surgically and to call attention to the fact that whereas the first 12 cases were operated upon without a death, there have been three deaths in the last 4 cases, the deaths being dependent upon the starved condition of the baby at the time of operation. This experience only serves to emphasize the importance of as early a diagnosis as possible in order that the surgical operative measures may be undertaken with the very greatest chance of the baby's recovery.

I operated during the past 8 years upon two babies who were thought to have a pyloric tumor and it was found at operation that they had no tumor. One case recovered from the exploration and is well to-day. One case died of some strange skin eruption unconnected with the operation. I have never operated upon a case of supposed pyloric spasm.

All the X-rays were taken by Dr. Walter Dodd of Boston, Mass.

CASE I.—Wales.¹² A patient of Dr. C. W. Townsend, of Boston, and Dr. West, of Newton Center, Mass. The operation was done by Dr. C. L. Scudder. A boy baby. Weighed at birth 10 pounds, 11 ounces. He was artificially fed and vomited shortly after birth. The vomitus was the milk taken at a feeding. The vomitus contained no bile. The vomiting was expulsive in character and occurred immediately after each feeding. He lost two pounds in weight before the operation. At operation tumor was seen and palpated at the pylorus. A posterior gastro-enterostomy was done when he was 14 days old. This is the youngest recorded recovery after gastro-enterostomy for congenital pyloric stenosis. He weighed 33 pounds plus when he was 3 years and 2 months old. His diet was that of a healthy child for this age. He has always been inclined to constipation. See Fig. 2 (1910).

Weight May 18, 1910, 44 pounds.

He is now eight years old and has had no trouble with digestion. He has always been perfectly well excepting that he is a slightly nervous child.

CASE II.—Larrabee.¹³ A patient of Dr. Ilsley, of Medford,

¹² First reported in the Boston Med. and Surg. Jour., December 14, 1905.

¹³ First reported in the Boston Med. and Surg. Jour., February 22, 1906

Mass. The operation was done by Dr. C. L. Scudder. A boy baby. He was breast fed. His weight at birth was $6\frac{3}{4}$ pounds. He vomited soon after birth and constantly until the operation on the 24th day. The vomiting occurred usually after several feedings and came in spurts. Gastric peristalsis was observed after each feeding. No tumor was felt. The child lost in weight and strength. At operation a tumor was seen and palpated at the pylorus. The operation was a posterior gastro-enterostomy. When he was three years and eight months old his general diet was milk, eggs, broths, never meat excepting chicken occasionally; bread, crackers, potatoes, Ralston food, cereals, peanut butter, butter, celery, turnips, fruit.

Weight, May 24, 1910, $37\frac{1}{4}$ pounds, when three years and eight months old. See Figs. 3, 4, 5, 6 (1910).

In February, 1913, a bismuth X-ray was taken of the child's stomach and he was apparently perfectly well. Bismuth leaves by the stoma. He is now eight years old. See Fig. 7 (1913).

CASE III.—Colby.¹⁴ Patient of Dr. J. L. Morse, of Boston, and Dr. Day, of Newburyport. The operation was done by Dr. C. L. Scudder. A boy baby. He began to vomit when he was 16 days old and the vomiting was constant until the operation on the twenty-second day. The material vomited was the milk taken. The vomitus contained no bile. The baby was breast fed. He had lost in weight. Gastric peristalsis was observed. A pyloric tumor was felt. At operation the tumor was seen and palpated at the pylorus. The operation was a posterior gastro-enterostomy. The child's bowels are never constipated.

When he was two years and five months old his diet consisted of milk, soft boiled eggs, steak, lamb, and broths, butter, bread, cereal, potatoes, macaroni, puddings and fruit. See Figs. 8, 9, 10 (1910).

In February, 1913, a bismuth X-ray was taken of the child's stomach, and he was apparently in perfect health. The bismuth leaves the stomach by the stoma. He is now seven years old. See Fig. 11 (1913).

CASE IV.—Stevens.¹⁵ A patient of Dr. Charles Putnam, of Boston. The operation was done by Dr. C. L. Scudder. A boy baby. The child was breast fed and artificially fed. He began to vomit when he was 14 days old. The material vomited was the milk taken. The vomitus contained no bile. Gastric peristalsis

¹⁴ Reported in the Boston Med. and Surg. Jour., August 6, 1908.

¹⁵ Reported in the Boston Med. and Surg. Jour., August 6, 1908.

FIG. 1.



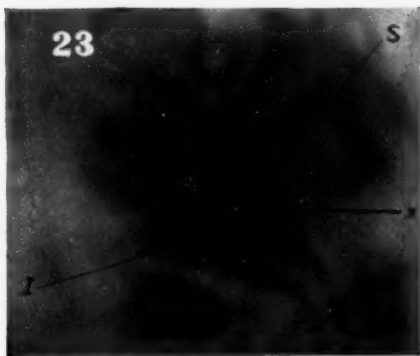
Normal adult stomach. X-ray after bismuth meal. Note that the duodenal cap and pyloric part of the stomach are to the right of the spine. X-ray by Dodd.

FIG. 2.



Wales. Case I. X-ray of stomach containing bismuth food 5 years following a posterior gastro-enterostomy. Note that the shadow of the bismuth is to the left of the median line. Bismuth is seen in the intestine. S, stomach; I intestine. 1910.

FIG. 3.



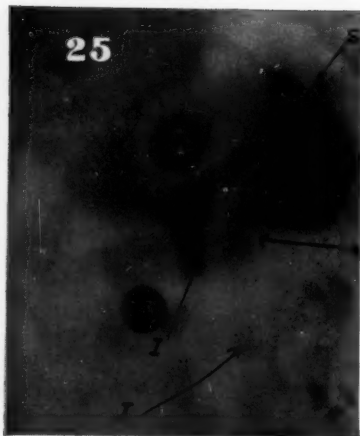
Larrabee. Case II. X-ray of stomach containing bismuth food $4\frac{3}{4}$ years following a posterior gastro-enterostomy. Note similar findings as those in the previous figure. S, stomach; I, intestine; X, stoma. 1910.

FIG. 4.



Larrabee. Case II. X-ray of stomach containing bismuth food $4\frac{3}{4}$ years following a posterior gastro-enterostomy. Note the definite stoma location and food seen coming through it. No shadow of food in the duodenum next to the pylorus. S, stomach; I, intestine; X, stoma. 1910.

FIG. 5.



Larrabee. Case II. X-ray of stomach containing bismuth food $4\frac{3}{4}$ years following a posterior gastro-enterostomy. Note the bismuth leaving the stoma at "X" the bismuth food in the intestine at "y." S, stomach; I, intestine. 1910.

FIG. 6.



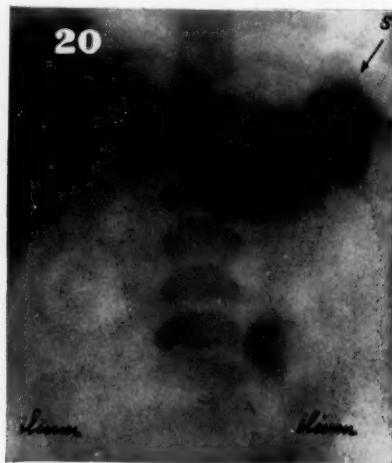
Larrabee. Case II. X-ray of stomach containing bismuth food $4\frac{3}{4}$ years following a posterior gastro-enterostomy. Note the bismuth has left the stomach; as faintly shown at "X" is the stoma. S, stomach; I, intestine. 1910.

FIG. 7



Larrabee. Case II. X-ray of stomach containing bismuth food eight years following a posterior gastro-enterostomy. 1913. No food seen going through pylorus. St, stomach; S, stoma; I, intestine.

FIG. 8.



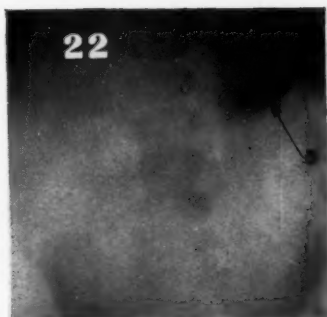
Colby. Case III. X-ray of stomach containing bismuth food $3\frac{1}{2}$ years following a posterior gastro-enterostomy. Note a later X-ray with more food in intestine and no shadow of food leaving the pylorus. S, stomach.

FIG. 9.



Colby. Case III. X-ray of stomach containing bismuth food $3\frac{1}{2}$ years following a posterior gastro-enterostomy. Note shadow of food in intestine and no shadow of food passing duodenum. S, stomach; I, intestine.

FIG. 10.



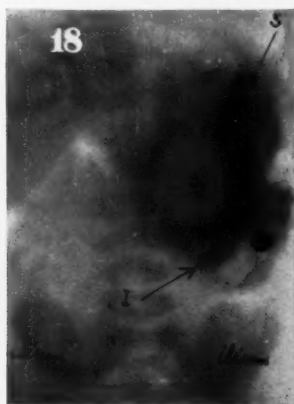
Colby. Case III. X-ray of stomach containing bismuth food $3\frac{1}{2}$ years following a posterior gastro-enterostomy. Note shadow wholly to the left of the median line. S, stomach.

FIG. 11.



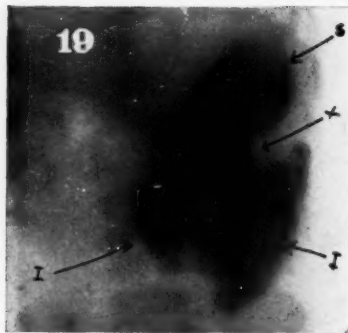
Colby. Case III. X-ray of stomach containing bismuth food seven years following a posterior gastro-enterostomy. 1913. Food not seen leaving pylorus.

FIG. 12.



Stevens. Case IV. X-ray of stomach containing bismuth food $2\frac{1}{2}$ years following a posterior gastro-enterostomy. Note shadows are all practically to the left of the median line. S, stomach; I, intestine. 1910.

FIG. 13.



Stevens. Case IV. X-ray of stomach containing bismuth food $2\frac{1}{2}$ years following a posterior gastro-enterostomy. Note food shadow at stoma "X" and in intestine below. S, stomach; I, intestine. 1910.

FIG. 14.



Stevens. Case IV. X-ray of stomach containing bismuth food five years following a posterior gastro-enterostomy. 1913. Note stoma at S.

FIG. 15.



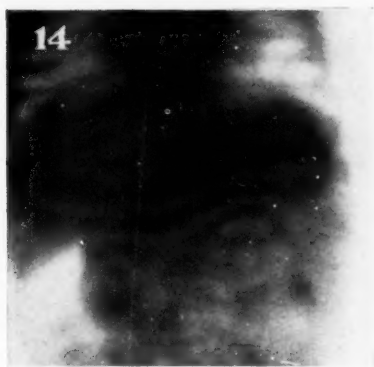
Nutting. Case V. X-ray of stomach containing bismuth food $1\frac{1}{2}$ years following a posterior gastro-enterostomy. Note no shadow at pylorus and duodenum, suggestion of stoma at "X."

FIG. 16.



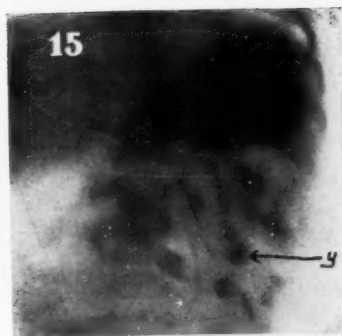
Nutting. Case V. X-ray of stomach containing bismuth food five years following a posterior gastro-enterostomy. 1913. Note food with bismuth leaving stomach at stoma S.

FIG. 17.



Matthews. Case VI. X-ray of stomach containing bismuth food, 1 1/4 years following a posterior gastro-enterostomy. Note shadow of food in stomach and intestine. Some shadow across median line to right side. 1910.

FIG. 18.



Matthews. Case VI. X-ray of stomach containing bismuth food 1 1/4 years following a posterior gastro-enterostomy. Note shadow limited to left side and intestine. 1910.

FIG. 20.



Hills. Case X. X-ray of stomach containing bismuth food three years following a posterior gastro-enterostomy. 1913. Food leaving stomach at stoma.

FIG. 19.



Matthews. Case VI. X-ray of stomach containing bismuth food, 1 1/4 years following a posterior gastro-enterostomy. Note shadow of food in intestine and stomach, none passing pylorus. Suggestion of stoma at "X." 1910.

F. G. 21.



Case XI. Note the stomach bulging the abdominal wall just under the left costal border, as it is contracting vigorously in attempting to overcome the obstruction at the pylorus, visible peristalsis. (Wilson.)

FIG. 22



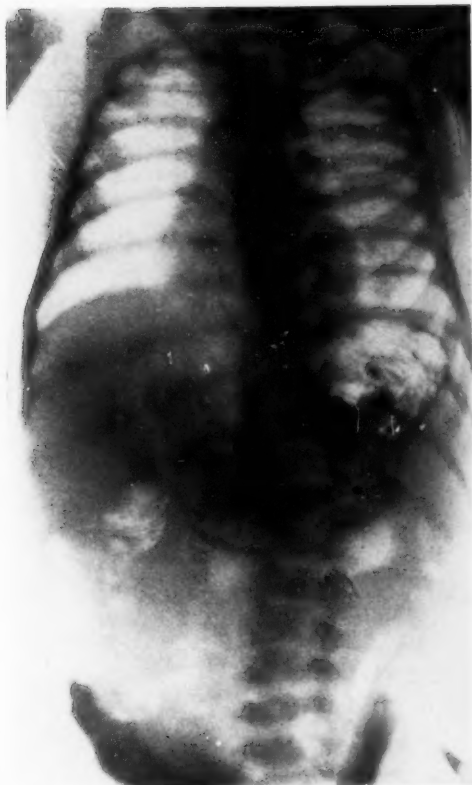
Case XI. Note that the visible peristaltic, gastric wave is more evident than in Fig. 21 as the wave passes further on the stomach toward the left. (Wilson.)

FIG. 23.



Case XI. X-ray taken after the operation showing the bismuth leaving the stomach through the new stoma. (Wilson.)

FIG. 24.



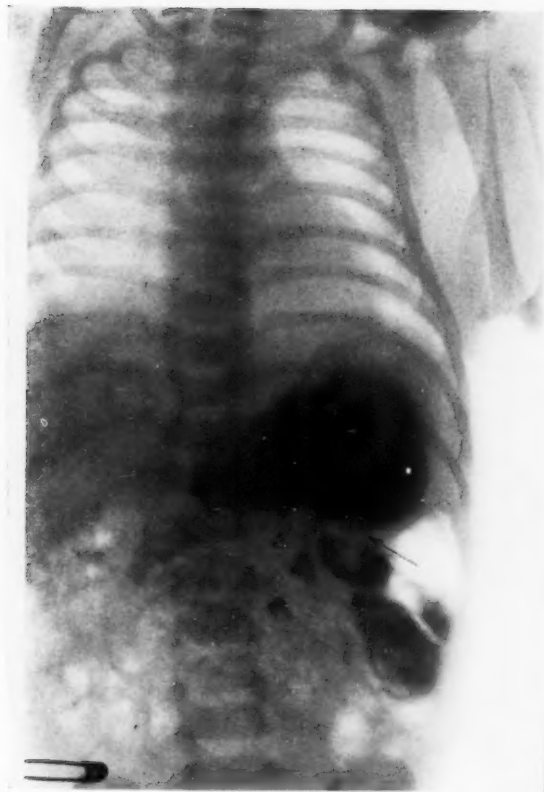
Case XII. An X-ray taken previous to operative treatment. Note the bismuth shadowy outline of the stomach and note that the bismuth remains largely in the stomach. The X-ray was taken some time after the injection of the bismuth. (Dunham.)

FIG 25.

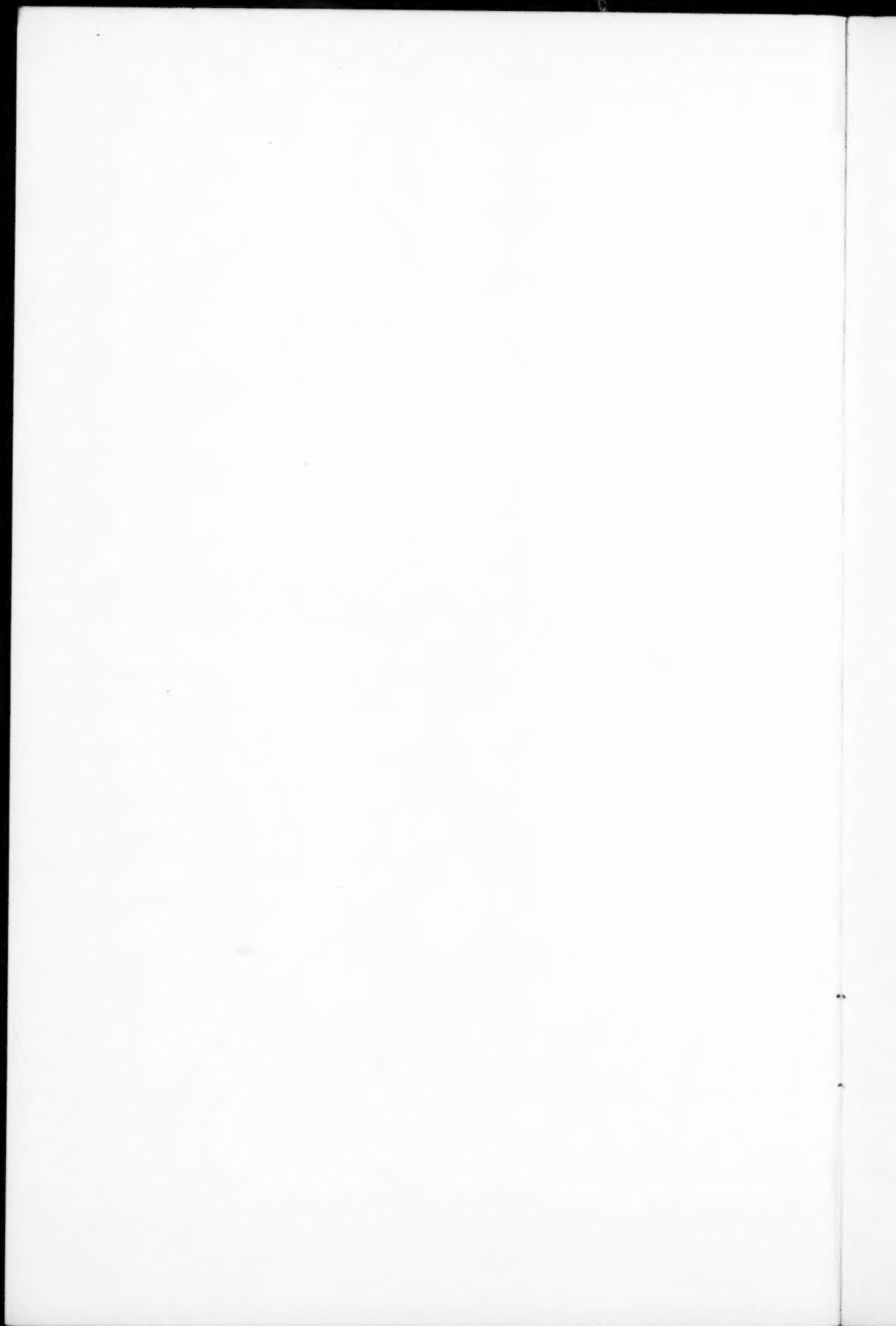


Case XII. X-ray taken after the operation. The bismuth is seen generally in the left side and middle of the abdomen after it has passed the gastro-intestinal stoma. (Dunham.)

FIG. 26.



Case XII. X-ray taken a little later than Fig. 25. Note the larger amount of bismuth in intestine and the indicated situation of the stoma (indicated by the arrow). (Dunham.)



was evident. No pyloric tumor was felt. The operation was done when he was 25 days old. At operation tumor was seen and palpated at pylorus. The operation was a posterior gastro-enterostomy. At 10 months old the child weighed $20\frac{1}{4}$ pounds. When he was two years and 4 months old he weighed $31\frac{1}{2}$ pounds. He was in perfect health. He is inclined to be constipated, and laxatives have to be used a good deal. See Figs. 12, 13 (1910).

In February, 1913, a bismuth X-ray was taken of the child's stomach, and he was apparently perfectly well. The bismuth leaves stomach by the stoma. He is now five years old. See Fig. 14 (1913).

CASE V.—Nutting.¹⁶ A patient of Dr. Cook, of Fitchburg, Mass., and Dr. John L. Morse, of Boston. The operation was done by Dr. C. L. Scudder. A boy baby. He began to vomit at 4 weeks and 5 days old. He weighed at that age $8\frac{1}{2}$ pounds. The movements were normal in color. The character of the vomiting was explosive. The vomitus consisted of the milk taken at a feeding. The vomitus contained no bile. There was very marked gastric peristalsis felt through the abdominal wall. A pyloric tumor was indistinctly felt in the region of the pylorus. At operation a tumor was seen and palpated at the pylorus. A posterior gastro-enterostomy was done when the child was 7 weeks and 5 days old. His weight at the time of the operation was 6 pounds and four ounces. At the end of three months after the operation he weighed $11\frac{1}{2}$ pounds. He seemed to be in perfect health.

The food given immediately after the operation was breast milk and a home modification. The food, May, 1910, consisted of a mixture of top milk, cream, peptogenic milk powder, lime water, and boiled water, 4 ounces, given every 2 hours, approximately.

Weight, May 29, 1910, 27 pounds. See Fig. 15 (1910).

In May, 1913, a bismuth X-ray was taken of the child's stomach and he was apparently perfectly well. The bismuth left the stomach by the stoma. He was four years and nine months old, and weighed 42 pounds. See Fig. 16 (1913).

CASE VI.—Matthews.¹⁷ A patient of Dr. Elam, of Gardner, Mass., and Dr. John L. Morse, of Boston. The operation was done by Dr. C. L. Scudder. A boy baby. He weighed 6 pounds

¹⁶ Reported in the Boston Med. and Surg. Jour., March 4, 1909.

¹⁷ Reported in the Boston Med. and Surg. Jour., Sept. 15, 1910.

at birth. He was breast fed and seemed in good health for the first 3 weeks. He increased in weight to 7 pounds and 12 ounces. When he was 3 weeks old he began to vomit about an hour after each feeding. The material vomited was the milk taken at a feeding. The vomitus contained no bile. The character of the vomiting became explosive. There was a loss in weight of one-half pound in the ten days previous to the operation. A pyloric tumor was easily palpable. There was marked gastric peristalsis. At operation a tumor was seen and palpated at the pylorus. A posterior gastro-enterostomy was done when the child was five weeks old.

Weight, May 18, 1910, 21 pounds. The food taken was breast milk. See Figs. 17, 18, 19 (1910).

In May, 1913, the child's mother reported that he was perfectly well excepting that he is subject to colds.

CASE VII.—D. H., Jr.¹⁸ A patient of Drs. Morse, Swain, and Dunn, of Boston. The operation was done by Dr. C. L. Scudder. A boy baby. His weight at birth was 9 pounds. He began to vomit when he was six weeks old. At that time he weighed 10 pounds and 11 oz. Breast feeding was discontinued immediately and artificial feeding begun. At the time of the operation he weighed 8 pounds and 11 oz. The vomitus contained specks of bile. The amount vomited was the whole of a feeding. The stools were meconium-like. Gastric peristalsis was visible. A characteristic tumor was palpable. At operation a tumor was seen and palpated at the pylorus. A posterior gastro-enterostomy was done at 7 weeks and 4 days of age. The child was in perfect health when he was 10 months and 24 days old, and weighed 20¼ pounds, May 18, 1910.

In May, 1913, the mother reported that the child was perfectly well as far as his digestion was concerned.

CASE VIII.—W. F. H., Jr.¹⁹ A patient of Dr. Straw, of Manchester, N. H., and of Dr. John L. Morse, of Boston. The operation was done by Dr. C. L. Scudder, April 22, 1910. A boy baby. He weighed 7½ pounds at birth. The baby was breast fed. He began to vomit when he was 16 days old. For the next two weeks he was fed a mixture of whey and modified milk, with a rectal feeding of peptonized milk every 2 hours. He had occasional cyanotic attacks. He had dilatation of the stomach, collapsed bowel and meconium-like movements. He had lost much flesh. His

¹⁸ Reported in *Surg., Gynec. and Obstetrics*, Sept., 1910, pp. 275-287.

¹⁹ Reported in *Surg., Gynec. and Obstetrics*, Sept., 1910, pp. 275-287.

general condition was poor. At operation a tumor was seen and palpated at the pylorus. The operation was done when he was 4 weeks and 4 days old. The operation was a posterior gastro-enterostomy.

This boy, Oct., 1913, 3 years after operation, is well and strong and developing normally.

CASE IX.—Gove. A patient of Dr. Charles A. Sturtevant, of Manchester, N. H., and Dr. John L. Morse, of Boston. The operation was done by Dr. C. L. Scudder, June 13, 1910. A boy baby. He weighed at birth $9\frac{1}{2}$ pounds. He was breast fed for five weeks and gained two pounds, but had colic all the time. Various formulas for feedings were then tried with indifferent success. He sometimes went several days without vomiting and then vomited several feedings at a time. The vomiting was occasionally explosive. He just held his weight. There was marked visible peristalsis. A mass about the size of a lead pencil could be felt just below the right border of the ribs nearly reaching the anterior axillary line. At operation a tumor was seen and palpated at the pylorus. A posterior gastro-enterostomy was done when the child was five months old. He is now, October, 1913, in perfect health, and is two years old.

CASE X.—Hills. A patient of Dr. MacLean, of Somerville, and of Dr. John L. Morse, of Boston. The operation was done by Dr. C. L. Scudder, September 11, 1910. A boy baby. He weighed four pounds at birth. The baby was breast fed. He began to vomit when he was thirty-five days old. Up to this time he had been well.

Certain things are interesting in connection with this case. First, the baby's vomiting began on or about the day that the mother's catamenial period was re-established. It was, therefore, a question whether it being a breast fed baby the mother's milk was altered in any way to cause the vomiting. Second, a definite tumor was felt in this case and the tumor was large and on the right side below the umbilicus. The tumor was low and felt so large that it was a question whether the baby might not be suffering from an intussusception. The stomach was known to be large. Third, the *vomiting existed only four days*, which is a very short time previous to treatment by operation, as these cases go. The reason for operating without the usual interval of medical experimentation is that there had been no let-up in the symptoms, and the pyloric tumor was distinctly palpable. The child was 39 days old the day of the operation.

The operation was a posterior gastro-enterostomy. The child recovered well from the anæsthetic and feeding was immediately begun with a mixture of brandy and water with 1 ounce of whey. The baby took his food well until about the tenth day, when there was some vomiting and spitting up of food, which was relieved by a change in the food formula. He gained weight constantly except for one set-back and weighed 6 pounds and 10 ounces November 8, 1910. He was born on August 3, 1910, and weighed 4 pounds. The baby is now, October, 1913, three years old and is well. See Fig. 20 (1913).

CASE XI.—Wilson. A patient referred to the Massachusetts General Hospital from the Out-Patient Department, April 7, 1911. The operation was done by Dr. C. L. Scudder. A girl baby (colored). The baby's weight at birth was $7\frac{1}{2}$ pounds. She was breast fed for 6 weeks but vomited continuously from the time she was two weeks old. The vomiting occurred at about 10 minutes after each feeding. There was marked gastric peristalsis visible after taking food. See Figs. 21 and 22. The character of the vomiting was explosive. At the time of the operation, April 7, 1911, when the baby was seven weeks old, she weighed 7 pounds and 13 ounces. A hard tumor was palpable at the time of the operation. A posterior gastro-enterostomy was done. Following the operation there was an immediate and steady gain in weight. At the time the baby was discharged from the Hospital, May 22, 1911, she weighed 8 pounds and 2 ounces, and was in good condition. See Fig. 23.

CASE XII.—Dunham. A patient of Dr. H. A. Chase, of Brockton, referred from the Out-Patient Department of the Massachusetts General Hospital to the House for operation. The operation was done by Dr. C. L. Scudder, April 25, 1911. A boy baby. The child was given 2 breast feedings after birth which were immediately vomited. He was artificially fed from that time. No food agreed well and the vomiting was projectile. There was no bile in the vomitus and no gastric peristalsis was visible. A tumor about the pylorus could be felt which appeared to be a hard mass the size of a pea. See Fig. 24. His weight at the time of the operation when he was four weeks old was 6 pounds and 7 ounces. The operation was a posterior gastro-enterostomy.

Following the operation he was given brandy in small doses, and after five hours he was given hourly feedings of milk which were retained. The baby made a steady gain. A second operation was done 24 days after the first operation for a slight hernia in

the scar. He was sent home on June 3, 1911, in good condition, weighing 8 pounds and 2 ounces. See Figs. 25 and 26.

CASE XIII.—Bonney. A patient of Dr. A. O. Sprague, of Turner Center, Maine. The operation was done by Dr. C. L. Scudder, on August 25, 1911. A boy baby. He weighed at birth $7\frac{1}{2}$ pounds. The baby was breast fed for three weeks, which was supplemented by bottle feeding. He gained one pound in weight. The stools were normal yellow. He took his food well and did not vomit.

The baby began to vomit about the time he was weaned, when he was three weeks old. The vomiting was projectile and occurred some time after a feeding so that occasionally several feedings were vomited at a time. The food was varied. The stools were meconium-like. The baby cried very little but did not sleep much. At the time of the operation his weight was 6 pounds. The child was poorly nourished and so emaciated that the skin hung in loose folds on the limbs. Gastric peristalsis was visible, and a hard tumor could be felt to the right of the median line. A posterior gastro-enterostomy was done August 25, 1911. Death occurred at 1.19 A.M., on August 26, from shock.

Autopsy.—The stomach and the seat of the anastomosis was removed. The tumor was characteristic. The stomach was slightly dilated and the stoma was patent. There was no evidence of peritonitis. Death was evidently due to shock.

CASE XIV.—Nyman. A patient of Dr. Nason, of Newburyport, and of Dr. J. L. Morse, of Boston. The operation was done by Dr. C. L. Scudder, October 14, 1911. A boy baby. His weight at birth in July, 1911, was 7 pounds and 14 ounces. He was breast fed, supplemented with modified milk every other feeding up to the time the baby was weaned. The movements were normal for the first few weeks and then became loose and green in color. The movements continued the same in spite of changes in food. In August he began to vomit following almost every feeding taken, and did not vomit again until the next feeding. The baby was pale and weak. The abdomen was somewhat distended and rather tense. There was no tenderness, masses or fluid made out. The liver was slightly enlarged. No tumor or peristalsis was noted during observations made at times almost hourly and at times just after feedings in the Childrens' Hospital. The stools were typical starvation stools. The vomitus varied from 3ss to 3ii in amount, and was watery with much mucus and curds; it was once

bile stained, and twice stained with "old blood." The character of the vomiting was explosive always. The baby lost 200 grammes in weight during the medical treatment in the hospital.

On October 14, 1911, when the baby was 11 weeks old, a posterior gastro-enterostomy was done. The child made a good recovery from the operation and did well. Two months later, on December 20, 1911, he weighed 14 pounds.

In January, 1913, the mother reported that the child had been well as far as his digestion was concerned.

CASE XV.—Lewis. A patient of Dr. C. H. Staples, of Malden, A boy baby. At birth, September 27, 1911, he weighed 7 pounds. He began to vomit October 28, 1911. There was some constipation although fecal material had always appeared in the dejections. The vomiting was explosive in character and occurred every half hour. There was no bile in vomitus. The baby lost rapidly in weight.

A posterior gastro-enterostomy was done on November 11, 1911, when the baby was about 6 weeks old. A large tumor was found at the pylorus. The stomach was very much distended and dilated. The baby did well immediately after the operation, although very feeble. He did not vomit and lived for 4 days. His death was due to his previous starvation and to shock caused by the operation.

The pathological specimen of the stomach and intestines showed the anastomosis in good condition; there was no peritonitis.

CASE XVI.—Monsen. A patient of Dr. Fritz B. Talbot, at the Massachusetts General Hospital. The operation was done by Dr. C. L. Scudder, July 29, 1912. A boy baby. His birth weight was 10 pounds. He was breast fed the first week of life and appeared normal, taking his feedings well. At the beginning of the second week he began to vomit everything taken, and vomited continuously up to the time of the operation when he was three weeks old. His weight at this time had reduced to 9 pounds. No definite mass was found in the abdomen but a firmness and hardness was apparent to the left of the epigastric region. At the operation a tumor was found at the pylorus and a posterior gastro-enterostomy was done. The baby did well following the operation and was breast fed. He was discharged September 3, 1912, in good condition, having gained 130 grammes in 11 days.

On April 25, 1913, the mother reported the child to be perfectly well. An X-ray of the stomach was taken with bismuth and the stoma was seen to be functioning. He was 10 months old and weighed 22 pounds. The baby is now, October, 1913, over a year old and is perfectly well.

CASE XVII.—Cohen. A patient of Dr. Fritz B. Talbot, at the Massachusetts General Hospital. The operation was done by Dr. C. L. Scudder, August 26, 1912. A boy baby. His birth weight was 7 pounds. The baby was breast fed the first three weeks of life and was apparently normal. The faeces were normal and there was no vomiting. At three weeks old he began to vomit directly after each feeding and continued to do so up to the time of the operation. The vomiting was projectile in character. A definite tumor could be felt in the epigastrium. There was a marked loss of weight. At the operation, when he was 5 weeks old, a tumor was found at the pylorus and a posterior gastro-enterostomy was done. The baby was very weak and in spite of stimulation he died at midnight after the operation, August 27, 1912. At the autopsy the tumor was found at the pylorus and the gastro-enterostomy was intact.

TABULATION OF THE CASES OF PYLORIC STENOSIS.

No.	Age at operation.	Duration symptoms.	Time since operation.	Post-operative X-rays.
1	14 days	14 days	8 years	5 years, 1910. Food through stoma
2	24 days	24 days	8 years	8 years, 1913. Food through stoma
3	22 days	6 days	7 years	7 years, 1913. Food through stoma
4	25 days	11 days	5 years	5 years, 1913. Food through stoma
5	7 weeks, 5 days	3 weeks	5 years	5 years, 1913. Food through stoma
6	5 weeks	2 weeks	4 years	1 year, 1910. Food through stoma
7	13 weeks, 4 days	11 days	4 years	
8	4 weeks, 4 days	2 wks, 2 days.	3 years	
9	5 months	5 months	3 years	3 years, 1913. Food through stoma
10	5 weeks, 4 days	4 days	3 years	3 years, 1913. Food through stoma
11	7 weeks	5 weeks		
12	4 weeks	4 weeks		
13	6 weeks	3 weeks	Died	
14	11 weeks	8 weeks	2 years	
15	6 weeks	2 weeks	Died	
16	14 days	14 days	1 year	1 year, 1913. Food through stoma
17	5 weeks	2 weeks	Died	

INTUSSUSCEPTION.*

REVIEW OF TWENTY-SEVEN CASES.

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THE patients with whom this brief review is concerned were under the care of various members of the medical and surgical staff of the Children's Hospital of Philadelphia and comprise all those recognized as intussusception in that institution. The first case was admitted in September, 1897, and the last one in February, 1913, the total number in these 17 years being 27.

In the histories of these patients there is no hint of any racial or hereditary predisposition. There are representatives of the Negro, Italian, Hebrew, Russian, Irish, and other unnamed nationalities. No family histories of interest were given.

A large majority of the patients were under one year of age. All except one were under 17 months, the oldest being 3 years old, and the youngest 3 months. Seventeen were boys and 10 were girls.

It is impossible to find any definite exciting cause for the production of the intussusception. It occurred only once in July and once in August, the season when summer diarrhoea is most common, and when the largest number of cases might be expected, if diarrhoea were an important factor in the etiology. There were 5 cases in June, 5 in October, 4 in February, and at least one case in each month throughout the year. There was only one in which the history indicates that indiscretion in diet might be held responsible: a child, sixteen months old, had been weaned two weeks before the acute symptoms began, and was being fed on milk, oatmeal, barley, and rice. In 15 cases mention is made of the kind of food the baby was taking, and of these 13 were breast fed. So commonly is the patient a nursling, though the history does not always record the fact,

* Read before the Philadelphia Academy of Surgery, November 3, 1913.

that it is accepted at the hospital as axiomatic that patients with intussusception are breast fed infants. The seasonal distribution and the preponderance of infants at the breast are rather striking in view of the common assumption that diarrhoea and irregularities and disturbances of peristalsis are largely concerned in the production of the condition. Usually the histories make no mention of symptoms preceding the acute attack; in one case it is definitely stated that there were none, one baby was subject to frequent coughs, five had diarrhoea for a few days to a few weeks, and two had more or less vomiting and diarrhoea for a month. These intestinal symptoms seem just as likely to have been due to the onset of the intussusception itself as to any preceding affection.

The acute symptoms which brought the patient to the hospital were vomiting and bloody stools. Although in two histories these symptoms are not recorded and in two others only one is mentioned, the combination is so constant as to be characteristic of the condition. The shortest period during which these symptoms were present was 12 hours, the longest 8 days. As is to be expected the duration of these typical acute symptoms has a distinct relation to the mortality rate, under both operative and non-operative treatment. With one questionable exception, death occurred in every case with a duration of four days or more—8 deaths in 9 patients—while of the patients in whom the symptoms existed one day or less, with one exception, all recovered—1 death in 7 patients—although a second one in this group succumbed later to a recurrence. In the intermediate group of 11 patients, where symptoms lasted 36 hours to 3 days, there were 6 deaths and 5 recoveries.

The examination for abdominal and rectal tumor is recorded in 24 histories. In 3, no mass could be felt in either situation; in a few it was found in one, but not the other. It is evident that the tumor might easily escape detection by abdominal examination if the abdominal wall were distended and rigid, as it sometimes was, and, of course, the rectal mass can only be found when the intussusception has progressed far enough to come within reach of the examining finger. There

does not seem to be any definite relationship between the extent of the invagination and the severity of the disease. It so happened that all the patients in whom no rectal tumor was found died, and only one where the abdominal mass was confined to the right side recovered. It is probable that the extent of the invaginated bowel depends entirely upon the underlying anatomical conditions, and has little to do with the amount of strangulation and destruction of tissue. Indeed, it seems probable that the extensive intussusceptions are those which form easily on account of the great mobility of the bowel and the large size of the colon, and for the same reasons are those most easily reduced.

Of these 27 children, 20 were operated upon and 7 were not, and it is rather surprising, at first sight, to find that operation gave a mortality of about 66 per cent., while non-operative treatment gave a mortality of less than 50 per cent. Only seven of the 20 operative cases recovered, while 4 recovered without operation and 3 died. In spite of this distinctly better statistical result for non-operative treatment, a more careful consideration will show, I think, that immediate operation is always the treatment of choice. As so often happens in acute abdominal disease, other means are exhausted before operation is undertaken and, in statistical reports, the failures of non-operative measures are glossed over by the ultimate result of operation. Among these 20 operative cases, 5 are definitely stated to have been treated by enemata unsuccessfully before operation was done, and all 5 died after operation. These should be considered as non-operative failures, and it is probable that there were several more, but the records are too incomplete to identify them. In one of these 5 cases, an anal protrusion was first noted 8 days before; the patient, a boy of 8 months, was then etherized and given an enema in an attempt to reduce the invagination. This treatment failing, the child was turned over to the surgeon for a second anæsthesia and laparotomy. Death occurred 5 hours after operation. In another one, it is said that the condition had existed 5 days and several enemata had been given before admission to the hospital. At operation the

small intestine was found to be necrotic at the entrance to the intussusception and was excised. The patient died 22 minutes after leaving the table. In a third case an enema had been given each day for four days with no result but blood. At operation a volvulus was found in addition to the intussusception, and the ileum was gangrenous and perforated. The child died 2 hours later. Such instances as these form no basis for a plea for even a preliminary trial of enemata or other non-operative measures, but urge strongly that laparotomy be performed at the earliest possible moment. It is practically certain that all the patients who died after operation would have died without it, and it is possible that some of those who died without it might have been saved by it. As the proper execution of the injection treatment requires a fairly long anæsthesia to allow the fluid to slowly distend the colon and force back the invaginated bowel and as the danger of recurrence after reduction is a very real one, it seems hardly necessary to argue in favor of opening the abdomen during the anæsthesia and of anchoring the ileocæcal region after reduction.

The histories do not give many details of the operations. In general the abdomen was opened in or near the median line, the tumor found, and reduction attempted by making pressure on its distal end, gently forcing the invaginated portion out of the proximal end, as paste is squeezed out of a tube.

The type of intussusception found is not clearly described in many of the histories. Usually the ileocæcal region was invaginated into the colon; in one case, at least, the ileum was invaginated into the cæcum through the ileocæcal valve. In several cases serious complications were present: once a double intussusception, once a volvulus, sometimes gangrene and perforation, requiring resection and anastomosis or the leaving of the damaged gut in the wound. All of these complicated cases died. When such complications were not present and the invagination could be completely reduced, the cæcum or ileum was sometimes anchored to prevent recurrence, and, at other times, the abdomen was closed without that precaution. One case illustrates the importance of such a procedure. The intus-

susception was easily reduced and no complications were present; the abdomen was closed without anchoring the bowel. The child did well for 4 days and had normal bowel movements. Then bloody mucus appeared in the stools and the tumor recurred. The abdomen was reopened and the invagination again reduced, this time with some difficulty on account of adhesions about the cæcum. The intestine was sutured to the abdominal wall and the wound closed. The patient again did well, but pneumonia developed and death occurred on the third day. The method of fastening the bowel to prevent recurrence varied; once the mesentery of the ileum was shortened, once an injected appendix was removed and its stump sutured to the abdominal wall, and in two other cases the peritoneum of the cæcum was sutured to the parietal peritoneum. These four patients recovered. The only fatality following an operation in which the history records this step was the case just quoted when the patient died after a second operation. This is good evidence that the procedure is not, in itself, detrimental.

Within the last month I have attempted to trace the 11 survivors of the 27 patients admitted to the hospital. Unfortunately I have not been able to find the 4 earliest ones. The other 7 have been uniformly free from symptoms of intussusception. The first was operated upon 5 years ago and has had excellent health ever since. The second was operated upon 2 years ago, developed an incisional hernia, which was repaired in another hospital a year and a half later, but has otherwise been perfectly well. The third was operated upon 17 months ago and has remained well since. The fourth had an operation 14 months ago and is still in good health. The fifth has remained well since the operation 13 months ago. The sixth passed through an attack of measles while in the hospital convalescing from operation and was well until whooping cough developed, of which the patient died 9 months after operation. The seventh was prepared for operation 8 months ago, but the symptoms having abated and the tumor disappeared, operation was held in abeyance. There has never been a recurrence and the patient is now in good health.

From a study of these histories and a few of the patients themselves, it seems to me that, when the diagnosis is made, there is only one treatment which is proper to pursue. That cases are cured by rectal injections is evident from this report and many others. But to secure a permanent cure by this means the reduction must be made neither too early nor too late. If made before the serous coat of the bowel is sufficiently inflamed to produce adhesions, recurrence is to be feared. On the other hand, if attempted too late, after the layers of the intestine are adherent to each other, or gangrene or perforation has occurred, the injection must necessarily fail, as illustrated by several cases in this series, and may do great damage. Immediate operation is always indicated. The only step in the operation which these cases suggest is in need of emphasis is the anchoring by suture of the ileo-cæcal region. The delayed cases with serious complications are almost hopeless from the start.

The mechanism of the production of infantile intussusception is not satisfactorily explained. The underlying factors which make the invagination possible are, of course, anatomical, and consist of unduly long mesenteries of ileum and cæcum and, perhaps, unusual disparity in the calibres of the small and large bowel, allowing the parts involved to move freely within wide limits and encouraging the entrance of the smaller into the larger segment. It is not hard to imagine that, having once entered the cæcum, the folded mass forming the apex of the invagination may be forced onward by colonic peristalsis. The difficulty is to understand how the invagination starts. The theories usually ascribe its beginning to some irritation which induces irregular or excessive peristaltic action. But there is little evidence in these 27 patients that such is the case. The increase in diarrhoeal diseases during summer does not in the least increase the incidence of intussusception and, with a few exceptions, the histories of these patients do not conform to the theory. My own impression is that the occasional preceding diarrhoea is a result, rather than the exciting cause of the trouble, and that the condition depends chiefly, if not entirely,

on the defective anatomical arrangement. And I have wondered whether the invagination does not start in the physiological pouting of the ileocaecal valve, which may become exaggerated, on account of the laxity of the attachments of the intestine, until it is caught in the grip of the colonic peristalsis and forced onward, dragging the cæcum with it. There is only a small proportion of cases in which the ileum is found invaginated through the valve, but it may easily retract after the bulkier head of the cæcum becomes the apex of the intussusception.

Whatever the precise mechanism be, it seems probable that the formation and spontaneous reduction of intussusception is much more frequent than our records show, and that the condition occurs many times in infants who die or recover without a correct diagnosis being made. Take, for example, a history like this: seven weeks before admission, vomiting and blood in the stools; then cessation of vomiting and bloody stools; 3 weeks later treated in the dispensary for gastro-enteritis, the child doing well; later a return of vomiting and bloody stools; then a protrusion from the anus. This was almost certainly an intussusception with spontaneous reduction and a later recurrence. Another patient had vomiting and blood in the stools a month before admission, these symptoms lasting 10 days and then abating; 3 days before admission vomiting and bloody stools recurred. How many infants have had vomiting and bloody stools due to a temporary intussusception which was spontaneously cured it is impossible to imagine. Here are two who temporarily recovered, but in whom a complete cure was not effected spontaneously. These instances are sufficient to show that the condition is not one which, having started, progresses until the patient dies or is cured by treatment, but rather suggest that the anatomical defect allows of frequent invagination and reduction until the inflammatory changes hold the gut in one or the other position. The condition is diagnosed and treated only when swelling and other inflammatory reactions prevent spontaneous reduction and cause more or less

obstruction of the fecal current and hemorrhage from the congested mucous membrane.

If it be so that cases of intussusception recover without intervention on our part, the cure must take place as a result of inflammatory adhesions which hold the parts approximately in their normal relations. The descriptions of the findings at operation and autopsy are too incomplete for definite conclusions, but there seem to be certain points at which the bowel remains folded upon itself longer than elsewhere—stations in the progress of the invagination—and at those points inflammation is most marked and adhesions are most likely to occur. The ileocæcal region is practically always involved. In the right-sided tumors the apex of the intussusceptum seems to be blocked by the hepatic flexure, and the inflammation is confined to the ileum, cæcum, and ascending colon. In those where the tumor runs transversely across the abdomen, the angulation at the entrance to the intussusception is in the neighborhood of the hepatic flexure, so that adhesions may form there. In the cases of left-sided tumor the angulation occurs at the splenic flexure, and adhesions may be expected there.

Assuming that some of these various forms undergo spontaneous reduction and permanent cure, it is interesting to speculate upon the conditions which may be present in after life. The mobile cæcum of the adult seems like a direct continuation of the conditions underlying infantile intussusception. If the lower portion of the ileum slips in and out of the ileocæcal valve under certain anatomical conditions in infancy, and is finally prevented from doing so by the formation of adhesions which its own pernicious activity have produced, we should expect to find in later life, within a few inches of the ileocæcal junction, bands of adhesions shortening the mesentery, and on either side of this area an unusually long mesentery permitting ptosis of the proximal ileum and the cæcum, with the production of a typical Lane's kink. I think intussusception offers also a plausible explanation for the obstructing inflammatory bands at the hepatic and splenic flexures. Mr. Lane explains the formation of these bands and kinks which bear his

name by an elaborate theory based on the reactions of the parts involved to the application of lines of force, and considers them the result of the ptosed condition of the intestines. It seems to me that it is more reasonable to assume that they are the result of some such process as I have indicated. I am not aware that Dr. Jackson, either, has included intussusception among the possible causes of the pericolic membrane he has described, but, if intussusception does recover, it seems almost inevitable that such a pericolitis should be one of its sequels. And, finally, a spontaneously cured cæcal invagination will account for some of the curious and almost impossible situations in which an appendix may be bound by old adhesions

MODERN LABORATORY METHODS IN THE DIAGNOSIS OF SURGICAL DISEASES OF THE GENITO-URINARY TRACT.*

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OF PHILADELPHIA, PA.

THE universal tendency in modern medicine is to rely more and more upon the assistance afforded by the laboratory in the prophylaxis, diagnosis, and treatment of disease, and of all the specialties surgery is perhaps the one most in need of this aid.

In those diseases of the genito-urinary tract either distinctly surgical at the beginning, or ultimately demanding such interference, much may be learned by the clinician from the microscopist. Every surgeon when confronted with a difficult case naturally welcomes every suggestion that will aid him in arriving at a diagnosis, and the purpose of this paper is to call attention to methods that are of distinct value if pursued with persistent care and careful attention to detail. The various pathological and bacteriological tests long ago accepted as conclusive, and concerning which there is no dispute, will not be referred to, but I wish to emphasize as strongly as possible the value of a properly conducted microscopical examination of the urine. In nearly every disease of the genito-urinary tract the urine offers to the trained eye more diagnostic information than can be learned by any single manifestation or any group of subjective symptoms. Carrying with it as it does unmistakable evidences of the source, severity, character, and comparative duration of the inflammation, there remains only the necessary time and care in its examination to yield definite and conclusive results. That a trust-worthy diagnosis may be made without previous knowledge of the age, sex, or clinical history of the patient is undoubtedly true, but ordinarily it is

* Read by invitation before the Philadelphia Academy of Surgery, November 3, 1913.

safer not to be too positive in the interpretation of the findings without some understanding of the symptoms exhibited. Only the modest and conservative claim is made that the methods to be described will invariably result in confirming a diagnosis already made, or in giving valuable aid to clear up a case presenting many elements of doubt.

Accompanying every inflammatory condition of the genito-urinary tract are varying numbers of pus-corpuscles and red blood-cells, and always to be seen with them are epithelia of various shapes and sizes, desquamated as the result of the inflammation and which never appear in normal urine. Upon the ability to differentiate these epithelia and to say with certainty from what part of the genito-urinary tract most of them are derived the location of the inflammatory disturbance is dependent. It is not proposed to enter into a long discussion as to the views of various authors on this disputed point. It is enough to say that the claims of Louis Heitzmann in his book on "Urinary Analysis and Diagnosis" are enthusiastically accepted by many specialists, and that these claims cannot successfully be refuted by those who offer only a refusal to acknowledge them, unaccompanied by scientific argument to sustain their opinion. Heitzmann's views are extensively quoted by most modern authors, and will be, must be, eventually concurred in by all.

By a long series of experiments, covering many thousands of cases, it has been found that epithelia of a certain shape and size appear in the urine at the inception and during the progress of certain inflammatory conditions. Careful study of the urinary sediment in cases where the clinical symptoms admitted of no doubt as to the diagnosis, led to the belief that the epithelia always found in the given disease were directly derived from the organ affected. Following these observations to their natural and logical conclusion it has been demonstrated that certain epithelia appear in certain inflammations and are never seen in normal urine. On such an argument the differentiation of urinary epithelia is based, and it should be as convincing and as susceptible of proof as the diagnosis of the clinician

in a case of pneumonia, where long observation of the symptoms manifested allow of no hesitancy in the verdict.

At this point it should be understood that the claim is not made to label by a specific name every single epithelium appearing in the urine, but any inflammation of sufficient intensity to produce clinical evidences will result in the desquamation of a number large enough to warrant positive opinion as to their source. Many cases naturally present the difficulty of multiple involvement, necessitating careful study, but the embarrassment, if temporary, is never unsurmountable, the comparative number of the various epithelia present offering a sure guide as to the organ most affected.

It is hardly necessary to state that of all the epithelia found in urine those derived from the kidney are of the greatest practical significance, and fortunately at the present time there exists no difference of opinion as to the certainty with which they may be recognized. Many authors persistently refused even this concession until the study of specimens obtained by ureteral catheterization proved conclusively that these epithelia, always of a definite shape and size, appear in all diseases of the kidney. From the convoluted tubules the shape is round or oval, and in size one-third larger in diameter than the pus-corpuscle. Of about the same size, only columnar in shape, are those derived from the straight collecting tubules, and their presence in moderate or large numbers is usually indicative of a severe inflammation.

By reason of their importance, and for convenience of description, surgical diseases of the kidneys will be considered first, and perhaps one of the commonest is calculus. According to Keyes calculus in the kidney or its pelvis is the most frequent cause of renal suppuration, but I cannot agree with this author when he adds that "catarrhal inflammation is not encountered with calculus." As is well known to the clinician the symptoms vary greatly—all the way from an almost complete absence of subjective signs to a combination of intense renal colic, pressure and reflex pains, hæmaturia, and finally, suppuration and abscess.

The importance of careful X-ray study must be given great emphasis, and of course a positive shadow offers reliable information as to the presence and location of the stone, but as about 75 per cent. of all calculi are composed of uric acid the findings are frequently negative or doubtful. Examination of the urine in these cases will often clear up the diagnosis, and the microscopical picture will vary with the extent and duration of the inflammation. Some observers lay great stress upon the presence of a moderate or large number of red blood-cells in the urine, but if the source of the trouble is to be positively determined they must be accompanied by characteristic epithelia from the kidney, and ureter. Crystals of uric acid gravel in large numbers, particularly the rarer forms of stellate and needle-like concretions, together with renal epithelia, pus-corpuscles, and red blood-cells, offer, if not positive evidence, at least a strong possibility of the presence of a stone.

Suppurative pyelitis caused by calculus in the pelvis of the kidney, may be diagnosed by the above features, accompanied by a large number of pus-corpuscles and the presence of a preponderating number of epithelia from the pelvis of the kidney and the ureter. The former (pelvic epithelia) are characterized by round, oval, or lenticular shapes, are much larger than those derived from the kidney, while smaller than those of bladder origin; and the latter (ureteral epithelia) are usually round and twice the diameter of the pus-corpuscles.

Strongly suspicious of calculus in the pelvis or an impacted stone in the ureter is the sudden change from features pointing markedly in that direction to an almost complete absence of the same. This calculous anuria is by no means uncommon, and the features shift from time to time as the urine flow is obstructed or released. I saw this beautifully illustrated not long ago in a case operated upon by a prominent surgeon of this city. I had the privilege of following the case for some time before operative interference was decided upon. Repeated X-ray examinations always produced a shadow on the left side three inches above the bladder, and the ureteral catheter was arrested at that point. The microscopical picture of a

possible calculus was not as pronounced as is sometimes depicted in these cases, but my diagnosis of calculus was based on the fact that the features appeared and disappeared at intervals, two specimens in one day showing totally different findings. The patient was operated upon, extensive incisions being made, the bladder opened, and no stone found. Three weeks later he had another attack of renal colic, returned to the hospital and acted as his own surgeon by passing a stone so large that meatotomy was necessary for its final delivery.

Another important factor in the diagnosis of renal calculus is the appearance of the epithelia when the pressure of a foreign body exerts itself, or hypertrophy of the organ affected is co-existent with other symptoms of inflammation. This pressure results in the production of so-called endogenous new formations or inflammatory corpuscles within the epithelia, and these formations are never present in large amount except in pressure of some kind.

Pyonephrosis of tubercular origin offers in the urine both macroscopic and microscopic points of diagnostic value. The urine is usually heavily turbid, and if allowed to stand the pus separates itself into a thick creamy layer at the bottom of the glass, the supernatant fluid being clear. This is in marked contrast to the persistently turbid appearance of urine voided in ulcerative cystitis, or the residual urine in a case of prostatic hypertrophy where the bacteria of decomposition will not allow of such a separation.

At first glance it would seem that the clinician should not long remain in doubt over a suspected case of renal tuberculosis, but there are many where the family and personal history, and the objective and subjective symptoms offer little or no help. Particularly difficult is the diagnosis between renal tuberculosis and an ascending colon bacillus infection. Often the only prominent feature is pyuria, and the laboratory is invoked to ascertain the cause. Search for the tubercle bacillus, while so often unproductive, often fails because the proper technic is not observed. The urinary sediment should be as concentrated as possible, obtained first by sedimentation, then by the

added power of the centrifuge, and many slides should be examined before the search is abandoned and the findings declared negative. The next procedure is usually the inoculation of a guinea pig, this of course frequently making the diagnosis positive, but even before these methods are indicated by the gravity of the case, microscopical examination of the urine will often yield valuable information, at least in pointing the finger of suspicion toward a possible tubercular process. Tubular casts are sometimes present, though not often, the chief features being a large number of pus-corpuscles, few red blood-cells, connective-tissue shreds, fat globules free in the field and studding the epithelia, and epithelia from the convoluted tubules, straight collecting tubules, and pelvis of the kidney.

A clue of great value is supplied by the appearance of the pus-corpuscles, which in a tubercular infection indicate with unfailing accuracy an impaired constitution. This diagnostic point was first announced and demonstrated by Carl Heitzmann and one has only to study a sufficient number of cases to be convinced of its soundness and practical value. Pus-corpuscles indicative of a good constitution appear in freshly voided urine as coarsely-granular, rather highly-refractive cells with no visible nucleus. As the constitution becomes impaired the granulation appears finer, the refraction diminishes, until finally the regular contour is lost, the edges are ragged, and one or more nuclei come into view. A combination in a given case of two or more of the varieties described, for example, a number of coarsely-granular, highly-refractive corpuscles in company with others of pale, finely-granular, irregularly-shaped, nucleated appearance would indicate an originally good constitution now impaired by disease. Because I have touched upon this point while discussing tuberculosis it must not be understood that its diagnostic value is applicable only to this disease. The same information is at our disposal in any inflammation of the genito-urinary tract of sufficient severity to produce pus-corpuscles in numbers large enough for comparative study.

Before leaving the subject of renal tuberculosis I wish to

refer with great emphasis to the extreme importance of obtaining by the ureteral catheter specimens from both kidneys so that both may be studied before surgical relief is attempted. The reasons are obvious, (1) to avoid the catastrophe of removing one kidney in the congenital absence of the other; (2) to ascertain positively whether the disease is unilateral or bilateral; (3) to estimate through the chemical and microscopical findings the degree of functional activity exhibited by one or both kidneys.

Estimation of the renal function has been attempted by means of chemical tests over and over again, each new process attracting for a time more or less enthusiastic attention, but one after the other all have been discarded as practically valueless. Cryoscopy, always cumbersome in the technic, has been proven entirely worthless, as in many cases where one kidney known to be badly diseased and the other performing the functions of both, it has been shown that the freezing-point varied little or not at all. Much was expected of the numerous forced elimination tests with urea, sodium chloride, water, and the dye-stuffs, but the consensus of opinion now is that they are of no value in estimating the functional activity of the kidneys. The injection of phloridzin, setting up an artificial diabetes, with the appearance of sugar in the urine in about one-half to one hour in normal kidneys, and its failure of elimination in nephritis indicates only that the renal function is somewhat disturbed, and the results are never uniform. More promising in its accuracy than any other is the phenolsulphonephthalein test, recently devised by Rowntree and Geraghty, but it can hardly be carried out by the general practitioner, and even in the laboratory involves the employment of much time and work to obtain results more easily arrived at in other ways. These tests have been briefly referred to only to be condemned, for it is difficult to understand why time should be wasted on them when microscopical examination affords such positive proof of all that we desire to know regarding renal sufficiency or insufficiency. As already stated the urine from each kidney must be collected by the ureteral

catheter, simultaneously and for the same period of time. Chemical and microscopical examination of the two specimens will indicate conclusively the extent and location of the disease, and the constitution of the patient being determined at the same time by a study of the pus-corpuscles, the surgeon has at his disposal all the information necessary to a prompt decision as to the advisability or contra-indication of operation.

Malignant disease of the kidneys may often be diagnosed by microscopical examination of the urine, and aids the surgeon considerably when the clinical symptoms are either vague, or confused by the severity of some co-existent infection.

Sarcoma may occur at any age, and at its inception, before the ulcerative process is established, is difficult of diagnosis. To admit of a positive opinion there must be present in the urine large masses or shreds of connective-tissue and the characteristic sarcoma corpuscles in large numbers. Connective-tissue in the urine does not receive the attention it deserves, probably because it is so often confounded with mucus or extraneous matters such as cotton and linen fibres. It consists of wavy, moderately-refractive fibres, having a tendency to form into bundles, and is found in ulcerative, suppurative, hemorrhagic, and traumatic inflammations. Especially marked in ulcerative processes of malignant origin, these shreds, filling as they sometimes do an entire field, and studded as they occasionally are with inflammatory corpuscles, are enough of themselves to warrant a diagnosis of malignant tumor. In combination with sarcoma corpuscles, which present the appearance of small, round, highly-refractive, even glistening cells, without nuclei, larger than red blood-corpuscles and smaller than pus-corpuscles, the diagnosis of sarcoma is positive.

Cancer of the kidney is difficult of diagnosis from the urinary findings alone, but when large masses of connective-tissue, filled with large multi-nucleated epithelia are seen, accompanied as sometimes occurs by typical cancer nests, the suspicion of cancer is usually confirmed by the ultimate clinical history of the case.

Surgical diseases of the bladder due to tumor are not sus-

ceptible of positive diagnosis until the ulcerative process has begun, but when desquamative shreds of the tumor are voided in the urine no difficulty should be experienced. What has been said of sarcoma and cancer of the kidney applies equally to similar growths in the bladder. Hæmaturia is one of the first and most prominent symptoms, and even before the disease has advanced to the stage where connective-tissue shreds, sarcoma and cancer corpuscles, and evidences of a chronic inflammatory process contribute to a positive conclusion the characteristic bladder epithelia are always present. With the exception of those from the vagina, bladder epithelia are the largest seen in urine. From the upper layer, a few of which appear in normal urine, the shape is the familiar pavement or squamous form. This changes to a spherical or oval contour (from the middle layer) when the inflammation becomes more intense, and the columnar variety (from the deepest layer) is the product of deep-seated infection or ulceration. In diseases of the bladder, as of the kidney, prostate, or any other part of the genito-urinary tract, the location of the inflammation and the ultimate diagnosis are absolutely dependent upon the differentiation of the epithelia always accompanying the other features in the case.

Papilloma of the bladder should especially be mentioned, because of its comparative frequency, and the striking microscopical evidences in the urine when this benign tumor is present. Hemorrhage, of course, is a prominent symptom, sometimes so profuse as to obscure more or less the other features, but rarely absent are the peculiarly-shaped connective-tissue shreds, once seen never forgotten, and of themselves almost pathognomonic. These shreds are very long, very irregular, having a tendency to coil or knob-like formations, and frequently contain fat globules or inflammatory corpuscles. With these features are pus-corpuscles and epithelia from the various layers of the bladder, particularly the columnar, many of them containing fat globules and the endogenous new formations indicative of pressure.

Intimately associated with the bladder is the prostate gland,

and diseases of this organ requiring surgical interference are common enough, and the diagnosis at times sufficiently obscure to demand whatever assistance the laboratory affords. Acute and chronic prostatitis, usually gonorrhœal in origin, seldom necessitates actual surgical aid, but abscess formation is of frequent occurrence and often goes unrecognized until rupture occurs. The diagnosis of such a condition is dependent upon the presence in the urine of a large number of pus-corpuscles, sometimes entirely filling the field, connective-tissue shreds, red blood-corpuscles, and epithelia from the prostate gland and its duct. These epithelia are about twice the size of pus-corpuscles, larger than those from the convoluted tubules of the kidney, and cannot be differentiated from those of ureteral origin, which are of the same shape and size. In prostatic abscess, however, the bladder and urethra are also involved, and the presence of epithelia characteristic of these organs will easily locate the inflammation, as in renal disease epithelia from the convoluted and straight collecting tubules and pelvis of the kidney enable us to eliminate the prostate as entering into the situation. The diagnosis of the majority of prostatic inflammations is rendered more simple by the presence in many cases of epithelia from the seminal vesicles and ejaculatory duct, but their surgical importance being negligible detailed description of them is omitted.

The urine in prostatic hypertrophy, especially of the senile type, presents another opportunity for positive diagnosis, oftentimes extremely valuable in hypertrophy of the so-called median lobe which has escaped the touch of the surgeon's examining finger. When the condition has reached the stage where urinary flow is obstructed and residual urine is always present, the bacteria of decomposition, of course, point strongly toward the prostate as being responsible. The epithelia from the prostate in such a case are always more or less filled with fat globules indicating chronicity, and endogenous new formations due to pressure of the enlarged gland. Epithelia from the neck of the bladder and those from the deeper layers of

the bladder itself are always present, as there is naturally an accompanying secondary cystitis.

It follows logically that this bacterial invasion and infection of the bladder cannot be long continued without an extension of the process through the ureters into the kidneys, and many cases of pyelonephritis are of such origin. This possibility, at times a dangerous complication of prostatic hypertrophy, necessitates careful study of the urine before operation is advised or attempted. Too many of these cases die shortly after operation, the mortality being ascribed to any but the real cause, *i.e.*, functional insufficiency of the kidneys. There should be no difficulty in making the diagnosis, and at the same time the surgeon is accurately informed as to the resistance apt to be exhibited by the patient.

The prostate is at times the seat of malignant disease, and such a diagnosis is made in the same manner as previously described when the kidney or the bladder becomes the host of this unwelcome visitor.

Stricture of the urethra presents a typical urinary picture, but is of no practical importance, as the clinical symptoms are clear, and routine examination by the surgeon leaves no doubt as to the diagnosis.

In conclusion I must ask your indulgence for the necessarily rough outline of the subject presented. Its importance is vital enough to deserve better and more detailed treatment, but I hope I have sufficiently accentuated the need of employing every modern laboratory test in the diagnosis of surgical diseases of genito-urinary origin, and the absolute necessity of determining before operation the functional power of the kidneys by microscopical examination of the urine.

A TECHNIC FOR PERFORMING A SHOCKLESS SUPRAPUBIC PROSTATECTOMY.

BY W. E. LOWER, M.D.,

OF CLEVELAND, OHIO.

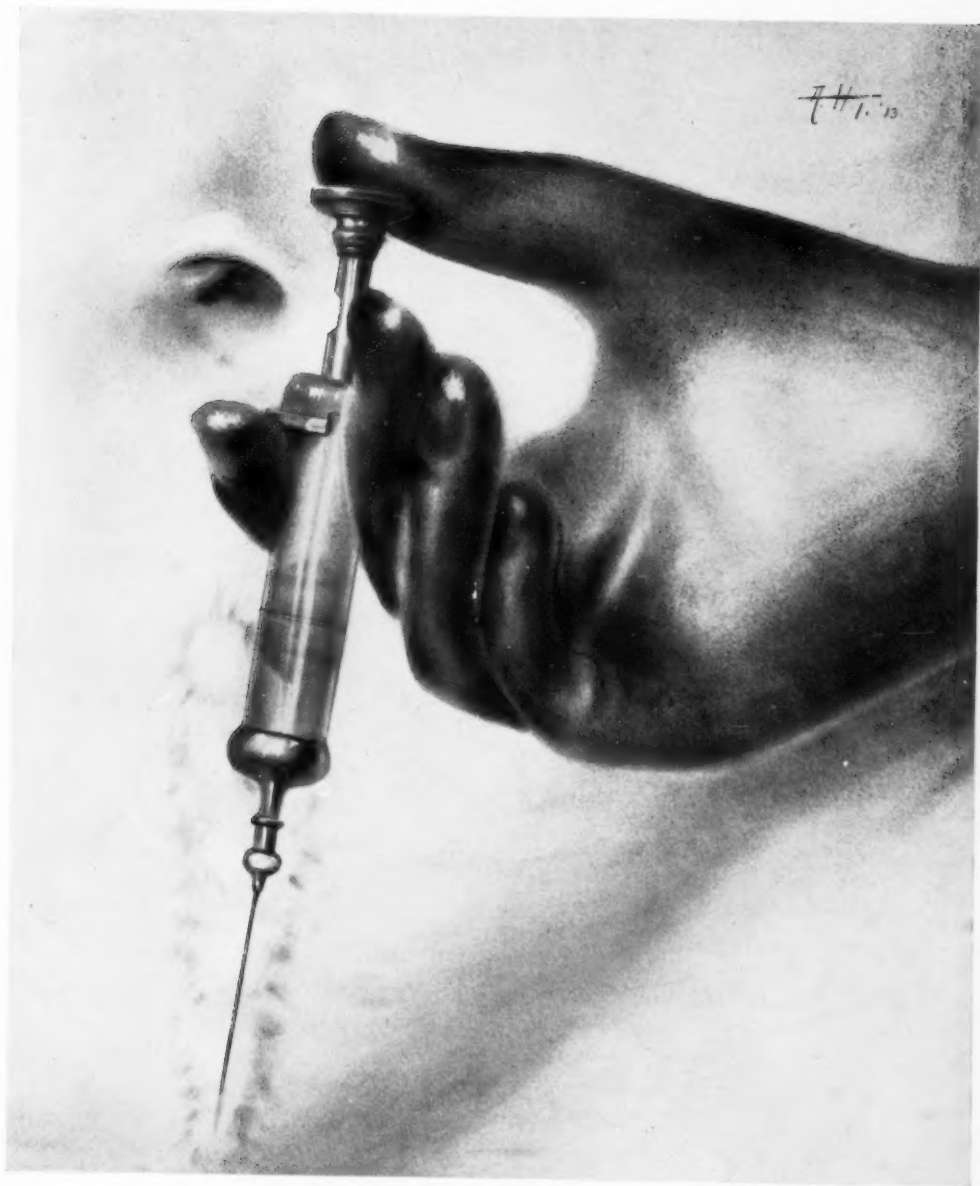
THE shock-producing factors of a prostatectomy are first, the effect of the anæsthetic; second, the *amount* of painful traumatism; and third, the hemorrhage. In so far as any of these factors can be minimized, post-operative shock will be lessened; if they can be eliminated then the operation becomes shockless and may be performed without hesitancy upon patients who, because of their age or because of diminished vitality from any cause, have been considered bad operative risks.

After a considerable experience and the trial of many different methods to diminish the dangers of this operation, the following technic has been evolved. Patients undergoing a prostatectomy performed by this technic are not only free from shock but are in splendid condition to combat any other untoward influence that may arise during convalescence.

TECHNIC.

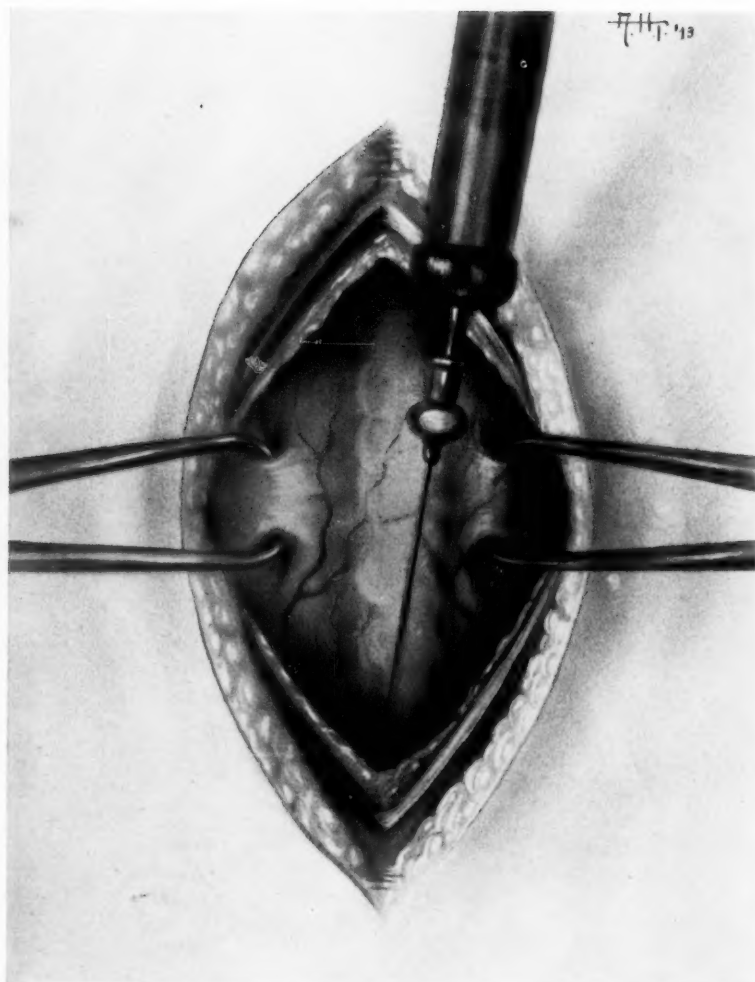
1. An hour before the operation the patient is given a hypodermic injection of morphine and scopolamine, the size of the dose depending upon his age and condition.
2. Immediately before the operation the bladder is irrigated and 60 to 90 c.c. of a 5 per cent. solution of alapin is injected through a catheter, the catheter is clamped, and both catheter and solution are allowed to remain.
3. Nitrous oxide-oxygen is administered by an expert anæsthetist; this anæsthetic when administered by one trained in its use being safer than ether and to some extent in itself a preventive of shock.
4. The bladder is approached in the usual way, except that the skin incision and every division of tissue is preceded by a thorough infiltration with novocaine in 1-400 solution (Fig. 1).

FIG. 1.



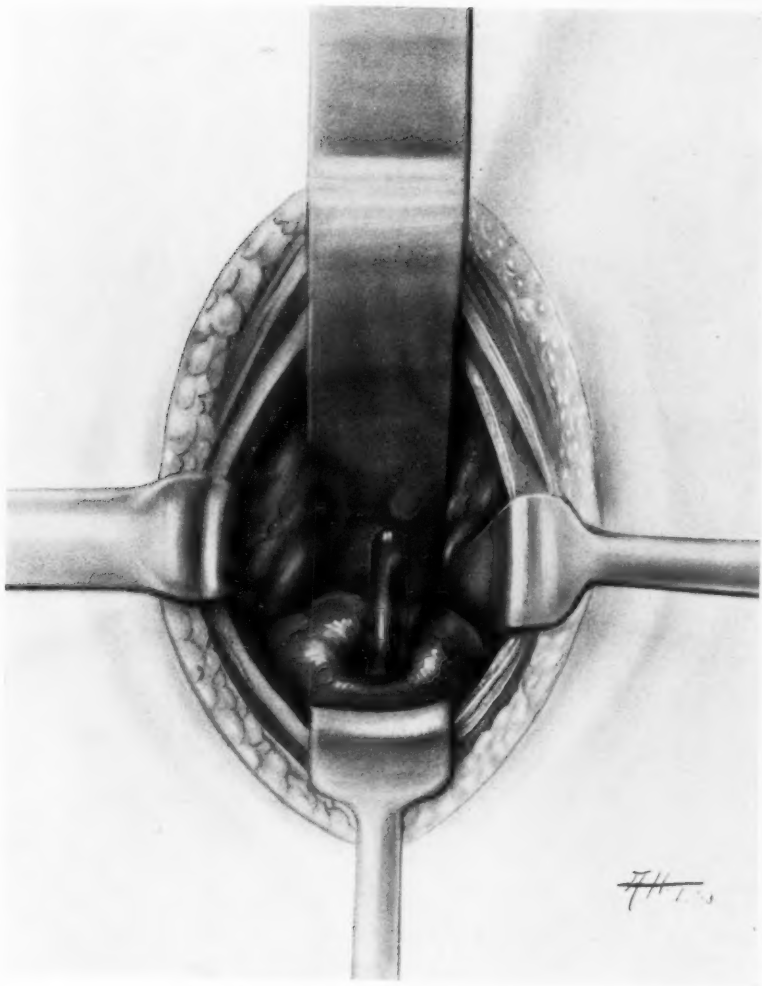
Infiltration of skin and fascia with novocaine.

FIG. 2.



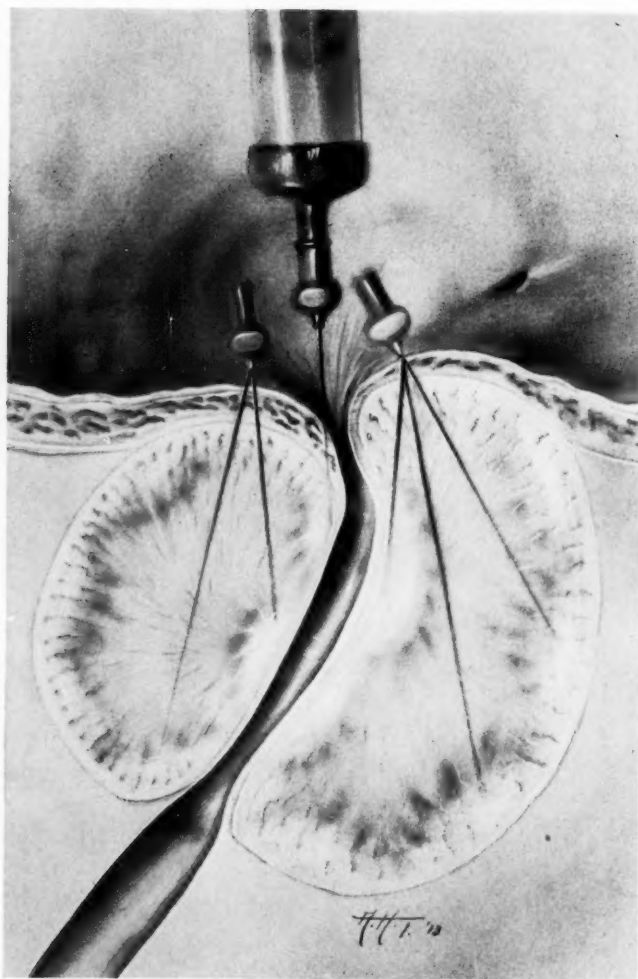
Infiltration of bladder wall with novocaine.

FIG. 3.



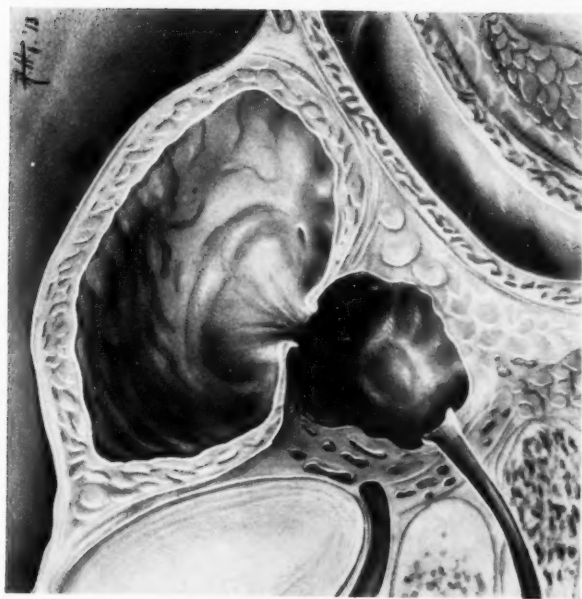
Intravesical exposure of prostate.

FIG. 4.



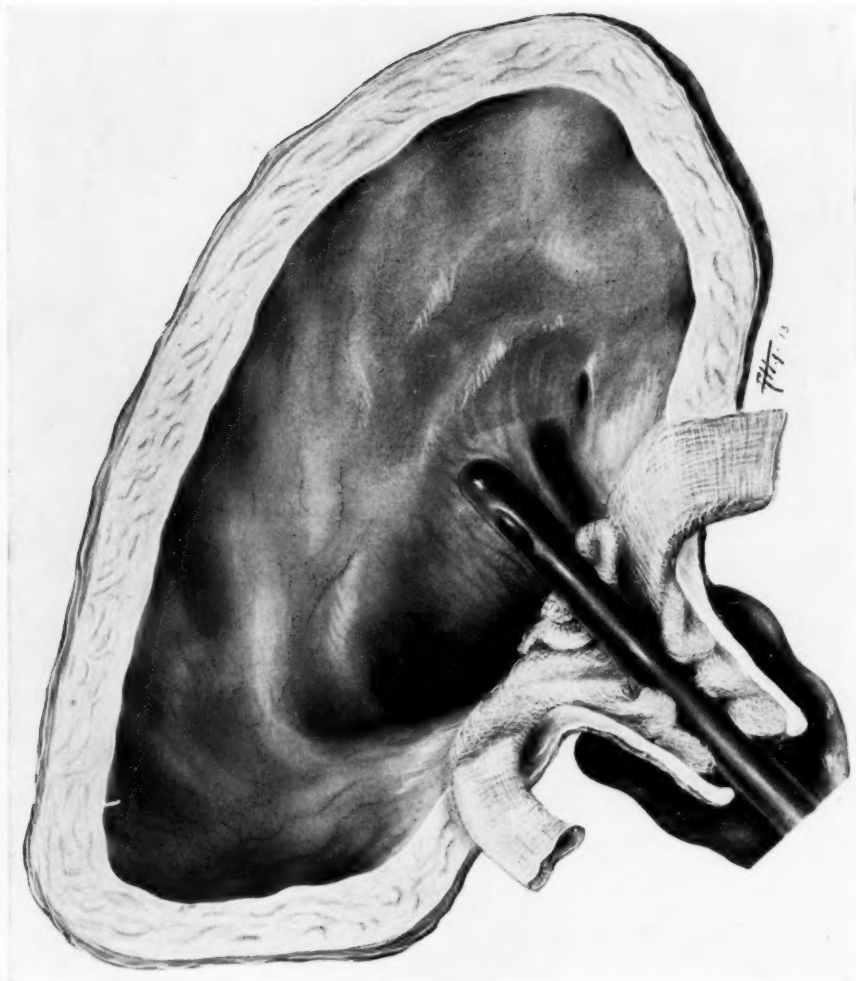
Deep infiltration along edge of capsule of prostate before removal.

FIG. 5.



Cavity left after enucleation of prostate.

FIG. 6.



Gauze packing by which the raw surfaces of capsule are brought in apposition.

5. When the bladder is exposed, it is elevated with curved bladder hooks and the bladder wall is thoroughly infiltrated with the novocaine solution (Fig. 2).

6. By gentle retraction and without injuring the cut edges of the bladder wall, the prostate is exposed intravesically (Fig. 3).

7. The bladder mucosa on the projecting prostate is infiltrated with novocaine, and along the edge of the capsule a deep infiltration is made (Fig. 4).

8. With careful and most gentle manipulations the prostate is enucleated with the finger (Fig. 5).

9. Narrow strips of gauze are packed along the side of the catheter on top of the mucous membrane so that the raw surfaces of the capsule are brought in apposition, a procedure which effectively prevents hemorrhage (Fig. 6). The two ends of the urethra are thus brought together, so that a continuous funnel-shaped mucous membrane is produced—a most important factor.

At the close of this operation the color of the patient will be good; the pulse and respiration will not be increased, in fact, may be even lower than before the operation. The patient will rest comfortably, will be free from nausea and mucus, can take water early, and a speedy, uninterrupted convalescence may be looked for.

TRANSACTIONS

OF THE

PHILADELPHIA ACADEMY OF SURGERY.

Stated Meeting, held November 3, 1913.

DR. JOHN H. GIBBON, Vice-President, in the Chair.

ARTHROPLASTY FOR ANKYLOSIS OF ANKLE.

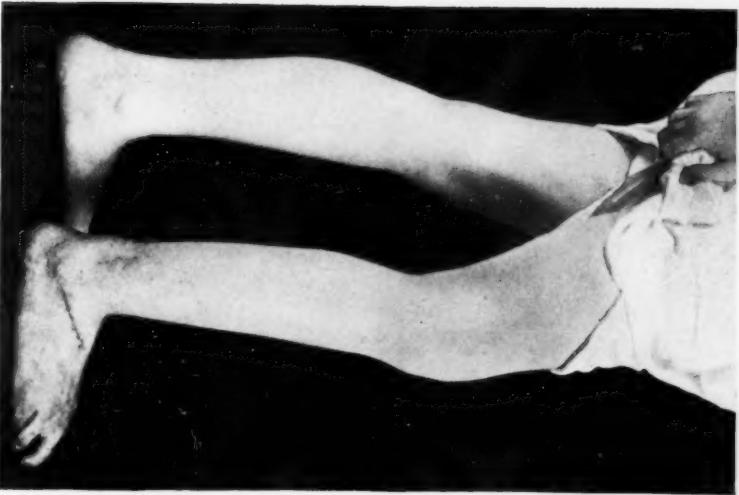
DR. ASTLEY P. C. ASHHURST presented a boy, aged eight years, who caught his right foot in the machinery of a grist mill, two years before coming under observation. Most of the soft parts on the inner side of the foot and ankle were torn away exposing the bones. Subsequently there was a great deal of sloughing, infection extended up the inner side of the leg nearly to the knee, and the boy was completely disabled for a long time. Part of the third toe was lost. On June 15, 1913 he was brought to the Orthopædic Hospital and admitted to Dr. Harte's service. The wounds had only recently healed; there were long scars densely adherent to the underlying bones all along the inner side of the foot and ankle, and up the inner surface of the leg almost to the knee. The foot was in a position of equinus, at 140 degrees with the leg, and there appeared to be bony ankylosis at the ankle (Fig. 1). This supposition was confirmed by a skiagraph, which showed ankylosis also of most of the tarsal bones, all bony outlines being obliterated (Fig. 4). The boy walked on the toes with the foot in a position of marked equinus and slight varus, and with the great toe in marked hallux valgus deformity (Fig. 5). There was nothing but scar tissue on the inner side of the foot and ankle, and this was densely adherent to the bone, absolutely no soft tissues being left. Careful and skilful massage was given for over seven weeks, but though some improvement occurred in the nutrition of the skin most of the cicatrices remained densely adherent to the bones. At length operation was decided on, and it was planned by Dr. Ashhurst to excise a wedge of bone of sufficient size to bring the foot up to

FIG. 1.



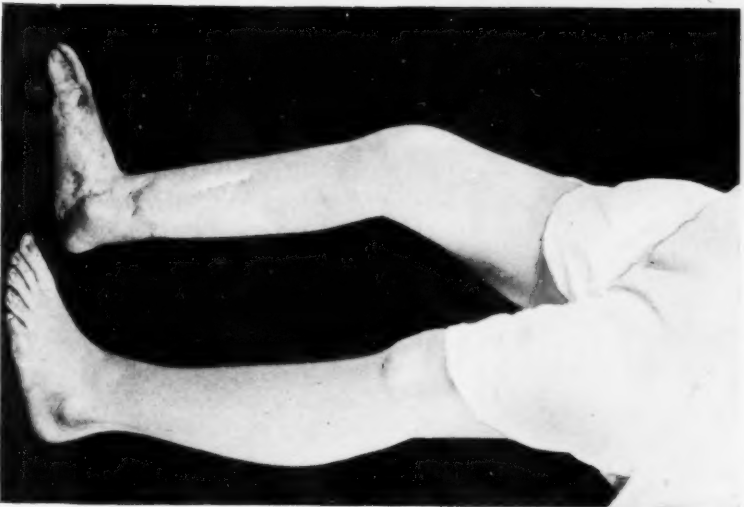
Bony ankylosis of ankle-joint in position of equinus (140°).

FIG. 2.



Showing external incision for arthroplasty of ankle.

FIG. 3.



Result of arthroplasty of ankle.

FIG. 4.



X-ray of ankylosis of ankle-joint in position of equinus.

FIG. 5.



X-ray showing traumatic hallux valgus, etc.

FIG. 6.



X-ray showing result of operation for hallux valgus.

FIG. 7.



X-ray showing lateral view of foot after arthroplasty of ankle-joint. In lateral view the new joint can be seen a short distance below the epiphyseal line of the tibia. In the anteroposterior view it can scarcely be seen.

a right angle with the leg, and then, if it had been found possible to preserve the lateral ligaments, so as to ensure stability of the foot on the leg, to transplant a piece of fascia lata between the bones, thus providing for motion at the ankle-joint. The loss of the subastragalar joint is much less disabling than that of the ankle-joint, and even in performing an arthrodesis of the ankle-joint in cases of infantile paralysis it is better to secure firm fibrous rather than actual bony ankylosis, so as to promote locomotion.

Operation, August 7, 1913: Dr. Ashhurst. Ether. Esmarch band above the knee. An incision was made on the outer side of the tarsus from below the external malleolus forward above the peroneal tendons as far as the extensor tendons (Fig. 2). This incision was carried to the bone, and all the soft parts were raised from the bones across the dorsum of the foot and ankle. Another incision, about an inch in length, was then made on the inner side of the ankle-joint, just in front of the internal malleolus and parallel to the shaft of the tibia. This incision had to pass through the old cicatrix adherent to the bone. The inner and outer wounds were then joined by burrowing from one to the other between the bones on the dorsum of the ankle and the overlying soft parts, thus raising all extensor tendons and the anterior tibial vessels out of harm's way. Next a wedge of bone was cut out by means of osteotome and gouge; the wedge had its base on the dorsum of the tarsus, and its apex at the posterior surface of what used to be the ankle-joint. After this wedge was removed, and the foot rendered movable on the leg, the next task was to excavate the tibio-fibular mortise. Then the foot came up to a right angle with the leg. Having preserved the lateral ligaments it was now found that there was only slight lateral mobility and tendency to valgus deformity (there had been varus deformity previously); therefore, it was determined to insert a flap of fascia lata so as to preserve the motions of the ankle-joint. Owing to the dense cicatrices all around the ankle and foot there was no possibility of employing a pedunculated flap. Accordingly an incision was made over the left thigh, and a piece of fascia lata and muscle was cut free at the point where the tensor fasciæ femoris is inserted. This transplant was two inches square and about one-quarter of an inch thick. The transplant was then placed in the new ankle-joint, and stitched in place with chromic

gut; there was no tendency for it to be displaced. The external incision at the ankle was closed in two layers, but the inner incision, through scar tissue previously adherent to the bone, permitted of closure only in a single layer of skin sutures. The operation took about one hour. The foot was dressed in plaster of Paris.

The plaster case was removed in two weeks, and the wounds were found healed. Some motion was possible in the ankle-joint. Three weeks after operation passive motion was begun; there was free voluntary motion of about 10 degrees, but the foot did not come up quite to a right angle with the leg. It might have been better to have lengthened the tendo Achillis at the first operation, but this was postponed because of the poor vitality of the tissues in which the arthroplasty was done.

Second operation, October 2, 1913 (eight weeks after the first operation): Dr. Ashhurst. The hallux valgus caused extreme deformity (Fig. 5), and it was planned to correct this as far as possible, and at the same time to lengthen the tendo Achillis so as to permit flexion of the ankle beyond 90 degrees. The old scar tissue was densely adherent to the projecting head of the metatarsal, but by turning up a flap with its convexity over the proximal phalanx good exposure of the metatarsal joint was secured. The head of the metatarsal was removed, and the toe brought around into proper position. The long extensor of the toe had sloughed away at the time of the original injury, and the only soft tissue which could be utilized to interpose between the sawn surface of the metatarsal and the base of the phalanx was the tendon of the abductor hallucis. This was accordingly turned into the new joint, and the soft parts closed. Then the remaining stump of the third toe was removed, the deforming cicatrix which covered it was excised, and an Agnew operation for webbed fingers was done to restore the contour of the toes as far as possible. The improvement in position is readily seen by comparing the X-rays made before and after operation (Figs. 5 and 6). Finally the tendo Achillis was lengthened by the usual Z operation. When the tendo Achillis had been divided it was found that free motion was possible in the ankle-joint: flexion to about 70 degrees, and extension to about 120 degrees. The foot was dressed in plaster of Paris at an angle of about 80 degrees with the leg.

The plaster case was removed October 23, three weeks after operation. There was a little sloughing of the margins of the flap over the metatarso-phalangeal joint of the great toe, but the other incisions were firmly healed, and the deformity was almost entirely overcome (Fig. 7). Passive motion was instituted, and the boy encouraged to walk around. Examination November 1, 1913, showed that he had free voluntary motion in the ankle from 85° to 95° ; and that passive motion was possible from 85° to 110° .

The patient was shown at this meeting, Dr. Ashhurst said, because he would leave for his home in Maryland before the next meeting. It is hoped that it will be possible to report improved motion at a future time.

RESULT OF EXCISION OF WRIST FOR TUBERCULOSIS.

DR. ASHHURST presented a man, twenty-eight years old, who came under his care in August, 1912. When four years of age he had suffered from tuberculosis of the right hip; a cold abscess formed and was opened, but healed soon. He was under treatment for the hip condition until the age of 8 years, being confined to bed 18 months, and on crutches for nearly 3 years. He eventually secured a very useful limb, with fair motion at the hip-joint, but with shortening and adduction, which caused a marked limp. The hip remains weak, and is subject to slight injuries. He walks on his toes.

In February, 1912 he fell and injured his left wrist, and was treated for five or six weeks on a splint for what was considered a fracture of the radius and two of the metacarpal bones. The wrist never became normal, but remained swollen and painful and perfectly useless. In June, 1912 he applied to the Surgical Dispensary of the Episcopal Hospital, and came under the care of Dr. Carmany, who recognized the true condition as tuberculous (Fig. 8), and dressed the wrist on a palmar splint. In August, when Dr. Ashhurst went on duty in the Dispensary, an attempt was made to secure more absolute immobilization by the use of a plaster-of-Paris splint, applied to the dorsum of the forearm and hand, with the wrist in slight hyperextension. Although temporary improvement took place for a few weeks, the disease then began to progress: the joint was hot, red, and painful; the entire carpus was puffed up, on the flexor and extensor surfaces;

the fingers were stiff and useless, and though the skin was in good condition and no sinuses were present, it was not considered safe to persist in conservative treatment, especially as the patient had another (healed) tuberculous lesion in the hip, and it was feared this might light up again. A skiagraph made at this time (early in September, 1912) showed involvement of the radius and ulna, all the carpal bones, and the bases of all the metacarpals, except that of the thumb. Unfortunately this plate was broken.

The patient was admitted to Dr. Frazier's service in the Episcopal Hospital.

Operation, September 6, 1912: Dr. Ashhurst. Ether. Esmarch band below elbow. The dorsum of the hand was split between the index and middle fingers, the incision extending on to the radius above the wrist, and being continued through the web of the fingers on to the palmar surface of the hand for about an inch. The extensor tendons were turned aside, the wrist-joint was opened, and the ends of the metacarpals cut off with osteotome; the ends of the radius and ulna were removed in like fashion. Most of the carpus was removed in one mass, but the unciform, the scaphoid and trapezium had to be removed piecemeal. The end of the thumb metacarpal was not cut off, as it appeared to be healthy. The synovial membrane, and the tendon sheaths on the flexor and extensor surfaces were all invaded by the granulatous tissue, and a rather tedious dissection was required to remove them. The Esmarch band was removed before any sutures were introduced, and there was very little bleeding except from one large branch of the radial which required ligation. The radius was then drilled in two places, and one drill hole was made in each of the metacarpals of the index and middle finger and a suture of aluminum bronze wire was used to approximate the hand to the radius, in the hope of securing firm bony ankylosis and thus arresting the disease. The soft parts were closed with chromic gut sutures; and a small drainage tube was left in the wound. The hand was dressed in almost full pronation, in slight extension, and fixed by anterior and posterior splints of gypsum. The time of the operation was an hour and a half.

Two days later the drainage tube was removed, without disturbing the deep dressings. The first dressing was made ten days

FIG. 8.



Tuberculosis of left wrist, June 7, 1912; duration 4 months.

FIG. 9.



Excision of wrist, 7 months after operation (March 29, 1913).

FIG. 10.



FIG. 11.



FIGS. 10 and 11.—Result of excision of wrist for tuberculosis—limits of supination and pronation.

after the operation, when the wound was found healed except at the point where it had been drained.

Subsequently a sinus formed on the anterior aspect of the wrist over the radius. This was dressed with mercurial ointment, and the hand was kept at rest in a gypsum splint. The sinus remained moist until the end of January, 1913, more than four months after operation; but during all this time the wrist was painless, and gave no evidence of active disease.

A light brace was now ordered, and when seen in March, 1913, the patient was regaining considerable use of his hand, and had fair strength in his fingers, and good thumb motion.

Examination, October 20, 1913, over a year since operation (Figs. 9, 10, 11): The patient keeps a cigar store, and has no discomfort in the wrist except on violent motion. He can lift and carry almost any weight with the arm extended, the force being applied in the long axis of the hand and forearm; but the hand is weak if force is applied at right angles to its long axis. He goes hunting, and uses his left hand to hold the gun, without difficulty. He can oppose his thumb to the index and middle fingers, but not to the ring and little fingers. His grip is strong. There is rotation in the forearm from a position of almost full pronation to beyond mid-supination (90°). There is slight hypæsthesia in the ulnar distribution to the fourth and fifth fingers. The hand inclines to the radial side, and the head of the ulna is prominent on the dorsum. There is scarcely any motion in the wrist-joint. The wire suture causes no symptoms, but appears to be palpable on the extensor surface of the wrist.

CONGENITAL DEFORMITY OF THE AURICLE AND EXTERNAL AUDITORY MEATUS, WITH LOP EAR.

DR. ASHHURST presented a boy, now eleven years old, with a congenital deformity of the left ear. Apart from the disfigurement produced by the lopping over of the pinna (Fig. 12) and the existence of supernumerary auricular cartilages on both sides of the head, the external auditory meatus was absent (Fig. 13). Dr. T. S. Stewart, skiagrapher to the Episcopal Hospital, who made an X-ray examination, thought the skiagraph showed no evidence of a middle ear. But Dr. C. C. Eves, aurist and

laryngologist to the hospital, who very kindly examined the boy, reported as follows:

"Yesterday I examined your patient, Elmer W., and demonstrated that without a doubt there is present a patent Eustachian tube on the left side. The Eustachian catheter was easily introduced into the opening of the tube, and by placing one end of the diagnostic tube into the depression of the auricle on the left side the familiar oscillating sound of the air entering the middle ear could easily be heard when the air was blown through the catheter. The oscillating sounds were so distinctly heard that I feel sure that he must also have an external bony auditory meatus. In testing his hearing on the left side I found that he could hear the higher pitch tuning fork for a short duration. Bone conduction on that side is increased. He also lateralizes for that side when the tuning fork is placed on the vertex.

"These tests indicate that his deafness is of an obstructive type which may easily be due to closure of the external auditory meatus by skin and cartilage."

The patient's birth had been secured by instrumental delivery, and the ptosis of the left eyelid is attributed to injury at this time. The parents also blame this as the cause of the deformity of the pinna, but in view of the other congenital deformities of the ear it seems more rational to consider the lop ear also a congenital deformity.

The boy was admitted to Dr. Frazier's service at the Episcopal Hospital, and on August 26, 1913, Dr. Ashhurst did the usual operation for lop ear, removing an area of skin from the back of the pinna and from a corresponding surface of the adjoining scalp, and suturing the ear back against the head with interrupted chromic gut sutures. The supernumerary auricular cartilages were also excised from both sides of the head. The result is shown in Fig. 14, from a photograph made a month later.

No operation was done at this time on the external auditory meatus, because the parents did not desire it, and it was not urged because Dr. Eves had not yet made his examination which demonstrated the probable existence of a middle ear.

IMPERFORATE RECTUM.

DR. ASHHURST presented a boy, nearly six years of age, who, on January 14, 1908, being then a baby fourteen days old, was

Fig. 12.



Lop ear, before operation.

Fig. 13.

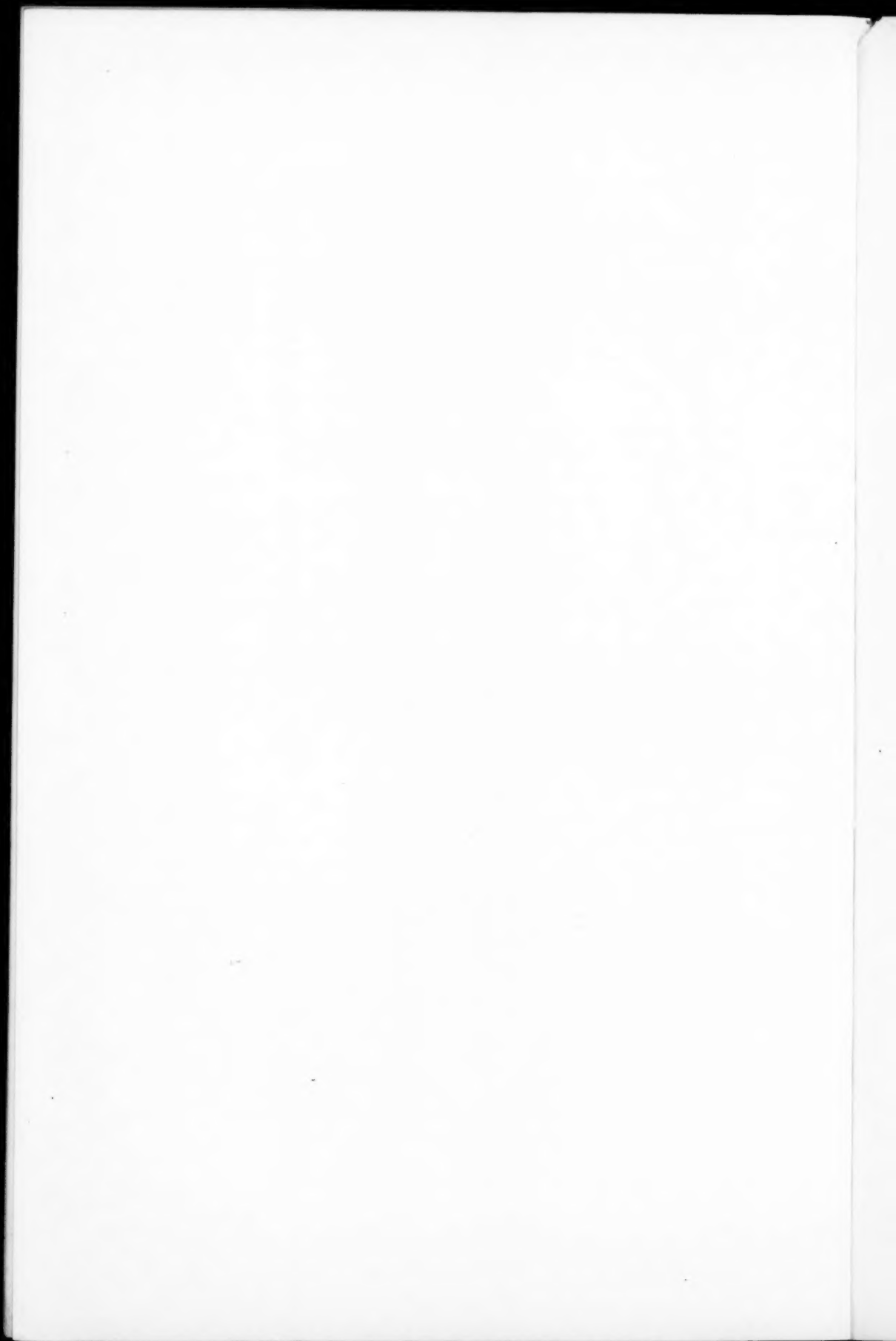


Congenital occlusion of external auditory meatus.

Fig. 14.



Lop ear, after operation.



brought to the Episcopal Hospital, and admitted to the service of Dr. G. G. Davis. *There had been no bowel movement since birth*; it was said that the urine once was very dark. The baby's abdomen was immensely distended, very tense, shiny red, and covered with many enlarged veins. The family physician had postponed sending the child to the hospital earlier, because a proctodæum was present. This, however, was found to be only three-quarters of an inch deep, ending in a blind pouch. The condition of the child was critical; the abdomen was so dreadfully tense that it seemed almost unsafe to bend the thighs up upon it for fear it would burst. However, the baby was placed on the operating table (lying on a hot water bag) in the lithotomy position. No anæsthetic was given. A median perineal incision was made 4 cm. in length, and deepened through the proctodæum to a depth of about 4 cm. from the skin surface. Here the rectal pouch was found, and incised; at once semisolid fæces squirted out in the form of a goose-quill or slate-pencil. The opening was dilated with forceps, and a large quantity of fæces was evacuated. The bowel (the mucosa of which was inflamed and red) was stitched to the skin with interrupted sutures of chromic gut. No ligatures were needed. The time of the operation was fifteen minutes.

The baby's bowels moved almost continuously for 12 hours after operation. No vomiting occurred after operation, the baby nursed well, and next day the abdomen was soft, not distended, and the redness and shininess had disappeared. Four days after operation the baby and its mother were sent home. Three weeks after operation, when the baby was five weeks old, the mother brought it to see Dr. Ashhurst at the Dispensary; the bowels acted normally, and the general health was excellent.

Nothing further was heard of the baby until he was brought to see Dr. Ashhurst again in September, 1912. He was now four years and a half old, and the complaint was that he had no control of his bowels.

Examination showed the anus and proctodæum as at birth, about half an inch deep, and ending in a blind pouch. Between this and the coccyx was a larger opening, about an inch in length, through which the fæces were passed. The mucocutaneous juncture of this opening appeared normal, but evidently there was no sphincter (Figs. 15 and 16).

Operation, September 30, 1912: Dr. Ashhurst (service of Dr. Frazier, at the Episcopal Hospital). Ether. Patient in Sims's position.

A grooved director was passed into the anal pouch, and was jabbed through into the rectum above (Fig. 15). The septum between the anal pouch and the rectal opening was then slit open,

FIG. 15.

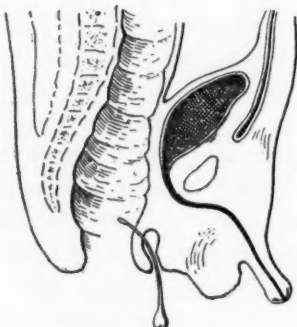
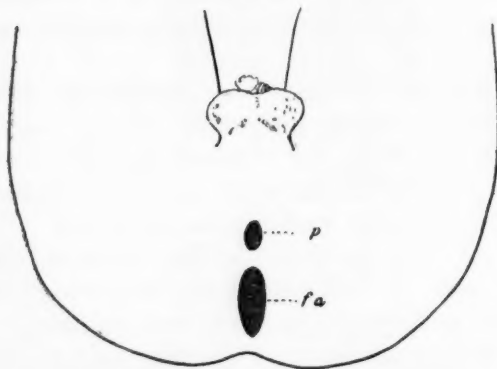


FIG. 16.

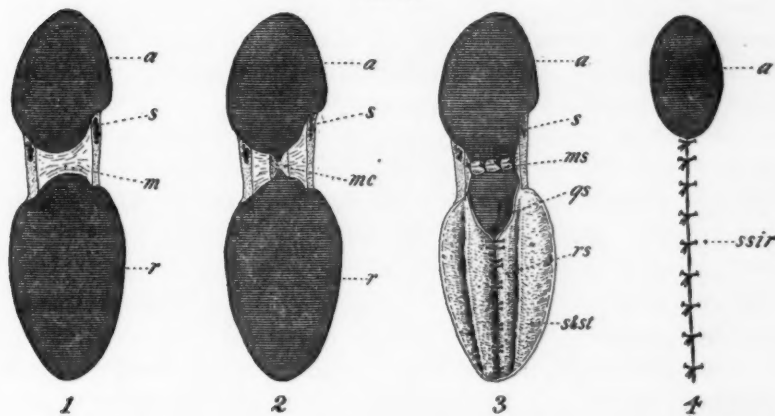


Figs. 15 and 16.—Proctoplasty for imperforate rectum, with secondary operation 4 years later to close a false anus in the perineum. *p*, proctodæum; *fa*, false anus.

on the grooved director as a guide. The fold of mucosa which remained on the anterior rectal wall (Fig. 17) corresponding to the septum between anus and rectum, was then divided in the long axis of the bowel, and was sutured transversely (Fig. 17, 2 and 3), thus restoring the anterior wall of the rectum. Then the rectal wall was dissected free from the skin of the perineum

all around the false anus and as far back as the coccyx, and was inverted toward the median line of the body in two flaps. These flaps were then united in the median line by numerous interrupted quilt sutures of No. 0 chromic catgut, all the knots being placed on the mucous surface of the rectum. This inverted the mucosa well (Fig. 17, 3). Then the ends of the sphincter ani (divided when the septum was slit open at first) were re-united. Next two buried sutures were employed to approximate the levator ani muscles between anus and coccyx; and finally, the skin between anus and coccyx was closed (Fig. 17, 4). A small tube was

FIG. 17.



1, *a*, anus; *s*, sphincter ani; *m*, mucous valve separating proctodæum from rectum; *r*, rectum. 2, *a*, anus; *s*, sphincter ani; *mc*, mucous valve cut; *r*, rectum. 3, *a*, anus; *s*, sphincter ani; *ms*, mucous valve sutured; *qs*, quilt sutures; *rs*, rectum sutured; *s* and *sl*, skin and subcutaneous tissues. 4, *a*, anus; *ssir*, skin sutured over inverted rectal wall.

left in the anal opening, to provide for passage of flatus, and if possible to relieve tension on the rectal flaps.

A few of the sutures sloughed out, but good control of the bowel movements was retained, and a finger in the anus detected a strong sphincter. When examined September 20, 1913, one year after operation, the anus was found normal, and perfect control of the bowels had been present ever since leaving the hospital.

EXCISION OF THE ENTIRE TONGUE FOR CARCINOMA.

DR. ASHHURST presented a man, aged fifty-nine years, who was an inveterate smoker. Apart from some trouble with his teeth in November, 1912, he had enjoyed excellent health of

late. One morning in April, 1913, on putting his pipe in his mouth (he always held it on the left side) he felt a burning and smarting sensation in his tongue. On his return to the house he looked in the mirror, and saw what appeared to be a slit on the left margin of the tongue, opposite the molar teeth. After one month he consulted his physician, Dr. H. M. Freas, who put him on mixed treatment, internally, and used iodine solution locally, over the alveolar border and the floor of the mouth, thinking the trouble arose in the teeth. Nevertheless an ulcer formed and increased in size; it was raised above the level of the surrounding tongue, and its margins and base were hard. Finally, about August, it was noticed that the ulcer was spreading up the anterior pillar of the fauces on the left side. Toward the middle of September, Dr. Freas thought some enlargement of the submaxillary lymph-nodes was occurring.

Dr. Freas brought his patient to see Dr. Ashhurst on September 20. The growth had then been noticed for about five months. The patient was a large healthy man weighing about 200 pounds. His blood-pressure was 80-160 mm. There were some albumen and casts in his urine, but the quantity of urine was sufficient, and the heart was normal. A "bronchial" cough had been present for the last forty years.

Examination of the mouth showed that the left border of the tongue from one inch back of the tip to the anterior pillar of the fauces was occupied by a raised, hard, ulcerated tumor, with sharply defined borders, and covered by an ashen gray slough. The mucosa covering the floor of the mouth was invaded, but the ulceration did not extend up on to the alveolus. The anterior pillar was just beginning to be invaded. The ulcer did not extend to the midline of the dorsum of the tongue. The tongue was not fixed. The floor of the mouth (mylohyoid) was not involved. Enlarged lymph-nodes were palpable on the left side at the level of the hyoid bone in the submaxillary region; no other lymph-nodes were palpable anywhere, on either side of the neck. A skiagraph showed no invasion of the mandible.

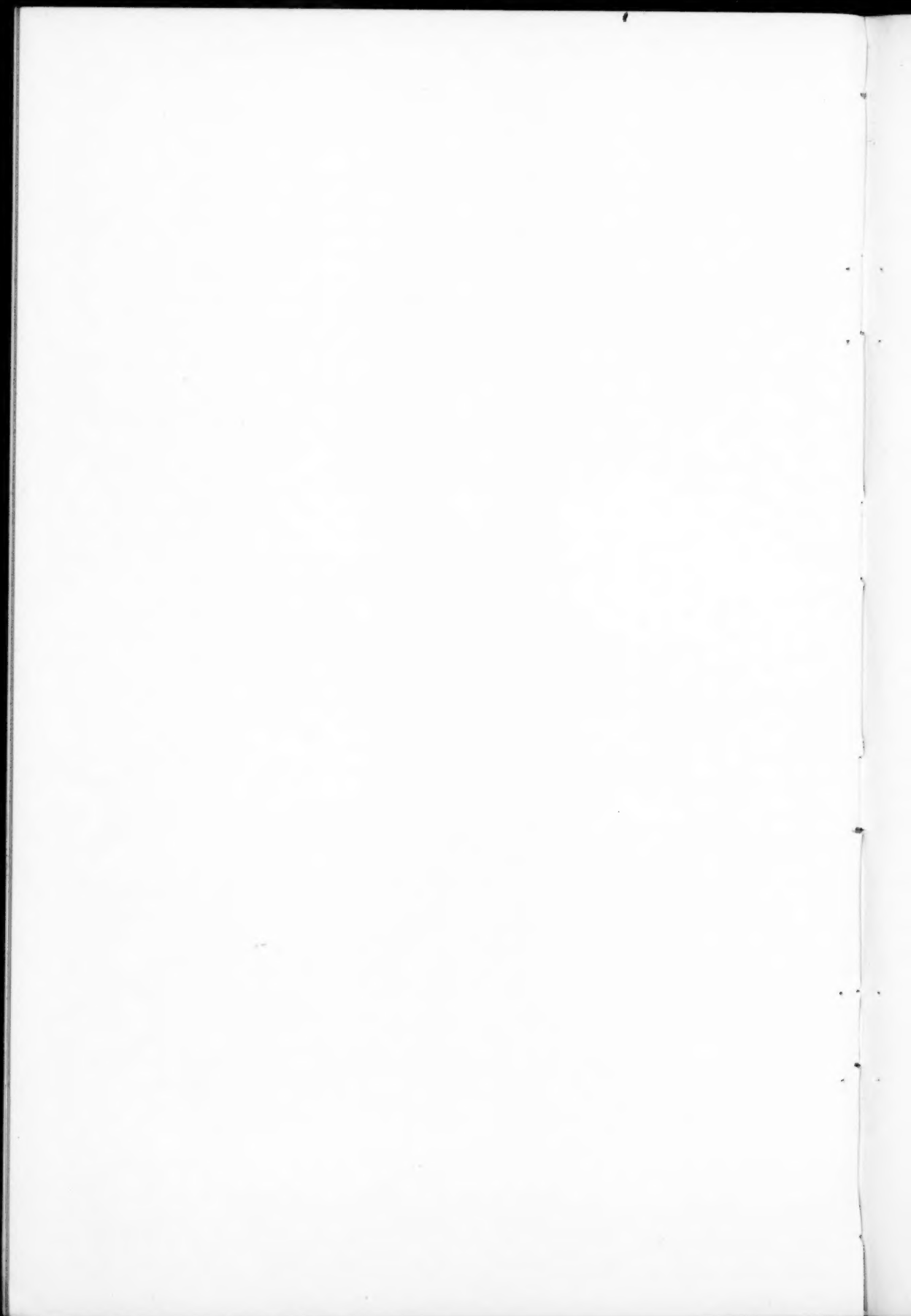
This was evidently a comparatively early case, as such cases go.

The patient was admitted to the Episcopal Hospital on September 23, and operation was done by Dr. Ashhurst on September 25, 1913, under anæsthesia by intratracheal insufflation of ether,

FIG. 18.



Tongue excised for carcinoma; submaxillary salivary gland and cervical lymph-nodes.



administered by Dr. W. E. Lee and Dr. Billings. Dr. Ashhurst said he would not go into the technic of the operation, as he hoped to bring this subject before the Academy on a future occasion. It sufficed to say that the plan of operation was modelled on Crespi and Bastianelli's modification of Langenbeck's method. The fat and lymphatics and the submaxillary salivary gland on the left side were removed in one mass, from the bifurcation of the carotid artery to the tongue and mastoid (Fig. 18); then the tongue was removed by turning aside the cheek. The neck wound was drained by a tube. The entire operation took two hours.

The patient left the table with a pulse less than 100, and subsequently it never exceeded this rate. There was no post-operative vomiting, and at no time any evidence of pulmonary irritation. The temperature averaged about 99° F., for the first few days, and then reached normal. The patient slept well the night after the operation, with a little paraldehyde, which he *said* tasted pleasant to him. Next morning he was able to make himself understood in talking. He was encouraged to swallow at once, and was able to take liquid nourishment from the first attempt. On the third, fourth and fifth days after operation a little liquid food discharged through the neck wound in swallowing, but this did not recur. He left his bed on the sixth day, and walked out of the hospital on the twelfth day after operation.

Swallowing of solid food was difficult for the first two weeks, but he found that if his mouth was filled absolutely full he could by effort force the mouthful back into the pharynx and so into the œsophagus. Before the end of three weeks he had dined with comfort off pork and beans, and took sauer kraut with relish. He talks with remarkable distinctness, considering the absence of his tongue, and is fairly well understood by the casual interlocutor, while his family never fail to understand what is said.

Pathological Report.—The specimens were examined by Dr. C. Y. White, pathologist to the Episcopal Hospital. He reports:

"The specimen shows a typical epithelioma of the tongue. The epitheliomatous tissue extends from the surface one-half to three-quarters of an inch into the meshes of the tongue. This degree of infiltration would indicate metastasis at least to the draining lymphatic glands. The lymphatic gland in the region of the sublingual salivary gland is free from metastasis. Numerous other microscopical sections,

from the root of the tongue and anterior pillar of the fauces, from the under surface of the tongue, and from the mass of tissue removed from the neck (submaxillary salivary gland, with submaxillary, submental, subparotid, and upper deep cervical lymph-nodes), fail to show any evidence of metastases. Microscopical sections made from the bone removed from the alveolus also are negative for metastasis."

DR. JOHN H. JOPSON said that in his experience the after history of cases of imperforate anus is marked not so much by incontinence as by obstruction from the contraction of the opening which is made to replace the imperforate anus. In one case under observation for between five and six years the condition was such that after three or four years operation was contemplated for the relief of the stenotic condition. In this case the anus was absent and the rectum was found high up; there was no proctodæum and the whole dissection had to be carried out through the perineum. Persistent dilatation was maintained up until a short time ago by means of English catheters and later by conical steel bougies. At one time the boy had distention of the colon as a result of stenosis and a plastic operation for its relief was contemplated, but always just as they would decide on this procedure he would improve and get all right; from last accounts he was in splendid health.

A short time ago he saw a child on whom he had operated eighteen months previously, the patient having been lost track of in the interim. At this visit the child was suffering from constipation, and he found here that there was a tendency to contraction of the septum which he had divided above the proctodæum, and he had to order dilatation in this case as well.

DR. EDWARD B. HODGE mentioned a case seen a few weeks ago, at three and a half years of age, on whom a colostomy had been done two days after birth by Dr. Warren Walker. In the paper he referred to Dr. Ashhurst brought out the fact that few colostomy patients lived to grow up and have the second operation, but this was a patient who did live and who was fairly well nourished. The child came into the Children's Hospital for the establishment of a rectum. Here also was a proctodæum.

The rectal pouch was easily reached at a depth of $1\frac{1}{2}$ inches, brought down and fastened to the skin with the addition of plastic work to secure a sphincter. So far the result has not been tested, as the colostomy opening has not yet been closed.

DR. JOHN H. GIBBON said that in his opinion the operation is

easier when done from below in cases of imperforate rectum because a dilated rectum is more easily found than after a colostomy has pulled it up and contracted it. He thought, however, it would be a mistake in these cases to try to do a plastic operation in the beginning; what Dr. Ashhurst did first, although only producing temporary relief, is the proper procedure.

One case of his own, operated on at the Pennsylvania Hospital later, came into the Jefferson Hospital with a very tight opening through which very little could pass and he found the rectum filled with densely hard enteroliths which bounced on the floor like marbles when removed with a scoop. He did a plastic but had to dilate the opening from time to time; the child now has perfect control.

INTUSSUSCEPTION.

DR. FRANCIS O. ALLEN gave a Review of Twenty-seven Cases of Intussusception at the Children's Hospital. For this paper see page 258.

DR. GEORGE G. ROSS said that he had seen three cases of intussusception. Two of these were in adults, the other in a young child about 22 months of age. The first adult case was one in which an intussusception occurred during convalescence from typhoid fever, giving rise to symptoms simulating those of perforation. On opening the abdomen an intussusception was found high up in the jejunum. It had occurred only a few hours previously, it was easily reduced, and the abdomen closed; the patient recovered. The second case was a man who had eaten deviled crab which was bad, he was taken violently ill that night; the following morning he had a violent pain in his abdomen and the doctor concluded he had obstruction of the bowel and hurried him to the hospital. He had an enterocolic intussusception which was tightly fixed, the ring about the intussusception was gangrenous, and the condition required an ileocolostomy. In addition to the distention from the obstruction there was a distention from the food poisoning, but the patient died. In the baby the condition came on without apparent exciting cause. He was crawling over the floor when he suddenly grabbed his abdomen and howled with pain. The diagnosis of intussusception had been made by the family doctor in the country. The child's abdomen was opened, an ileocolic intussusception was found, easily reduced and

stayed reduced, but during the trip to the hospital the child developed a cold and died 18 days after operation from pneumonia.

He was interested in the theory of intussusception causing the Jackson membranes or Lane's kink adhesions. He had felt that the vast majority of these conditions were acquired and that they must be due to some low grade inflammation of the peritoneum. Just what the most common cause is he had not been able to find out; although he had felt for some time that the appendix was responsible for a great many of them. A localized peritonitis of low grade without perforation of the appendix would seem a plausible explanation. He could understand how an intussusception which has been reduced and which stays reduced may produce a slow forming adhesion, the result of a low grade of localized peritonitis.

DR. WALTER ESTELL LEE said that in the sixth case reported he was the operator, and he could corroborate Dr. Allen's feeling that many of these intussusceptions relieve themselves. This child was seen by Dr. Howard Carpenter three days before operation, with symptoms of acute obstruction, and he advised it being sent to hospital but the parents refused. The next morning the child seemed perfectly well, the bowels moved normally and continued to do so for 48 hours, then the previous symptoms suddenly recurred and the child was brought to the hospital. At the operation the intussusception was very easily overcome, with the slightest traction the bowel was restored, and it was then sutured to the parietal peritoneum.

INJURIES TO THE ACROMION PROCESS.

BERNHARD MENCKE (by invitation) presented a paper with the above title for which see page 233.

DR. T. TURNER THOMAS said that if there is one thing about injuries to the shoulder that he would be glad to aid in establishing it is the importance of hyperabduction. It is to the shoulder what the twist of the foot is to the ankle. The great mass of injuries in the ankle region are due in the main to the turning of the foot inward or outward. That is not so obvious in connection with hyperabduction of the shoulder because the limb practically never remains in the position to which it is forced because gravity draws it down again.

If the arm is carried into abduction it is resisted first by the

capsule, that is the capsule offers the inelastic resistance and gives way first because it is to the skeleton at the shoulder what the bone is to the skeleton between the joints. In adults it is weaker than the bones, in children it is stronger, as shown by the relative frequency of dislocations of the shoulder and fractures of the clavicle in adults and children. When the arm goes into abduction the capsule binds and turns the scapula outward; it reaches a point where it cannot go further and when abduction is carried beyond that point something breaks and it is the capsule in the great majority of cases. When it tears it constitutes a break in the skeleton at the joint which means either a sprain or a dislocation, the sprain being a tear or break in the skeleton at the joint without displacement, and dislocation with displacement. In most cases the capsule tears. It is after the capsule tears that this contact takes place, that is the leverage and fulcrum effect from the contact of the humerus with the acromion, and it is right there occurs the crucial movement in these injuries to the shoulder region.

Hyperabduction is responsible not only for dislocations of the shoulder, but perhaps also for other conditions such as fracture of the surgical neck or a break in the lever at the fulcrum, fracture of the acromion, or a break in the fulcrum, and the upward dislocation of the outer end of the clavicle, the articulating surface of which favors the forcing inward, by the lever, of the acromion under the clavicle. Nothing is more difficult to prove than the actual mechanism of injuries to the skeleton.

MODERN LABORATORY METHODS IN THE DIAGNOSIS OF SURGICAL DISEASES OF THE GENITO-URINARY TRACT.

DR. A. T. GAILLARD (by invitation) presented a paper dealing with the above title for which see page 267.

DR. B. A. THOMAS said that he was not as enthusiastic as Dr. Gaillard over the value of the microscope in diagnosis of diseases of the genito-urinary tract, although giving it due credit for its great worth. Other procedures can aid in the diagnosis, and many of them are of more value than is the microscope alone. Dr. Gaillard said the radiogram left in doubt about 75 per cent. of diagnoses of calculus of the kidney, and that 50 to 75 per cent. are due to uric acid. It had been his experience that radiography will definitely determine renal or ureteral calculi, if present, in

at least 95 per cent. of cases, and we have never yet, with the best radiogram obtainable, found it impossible to make a correct diagnosis of stone in the kidney or ureter. In the case of a very soft urate, assuredly, the skiagram might not show the lesion. In his opinion the skiagram is the measure of greatest value in diagnosis of calculus of the kidney or ureter. Moreover, he could not see that urinalysis alone will diagnose the lesion so far as calculus is concerned, whether of the kidney parenchyma or of the pelvis or ureter when judged from the cytology; it may suffice to locate the inflammatory site, but does not specify that the real lesion is calculus. The cystoscope cannot be superseded by cytological examination in the diagnosis of many of these conditions, particularly in lesions of the bladder, where it is better for the patient to make a definite diagnosis by the cystoscope than to subject him to the lengthy process and uncertainty of repeated urinary examinations. Then again with all due respect to cytology of the urine, it is impossible except by making serial sections of certain tumors of the bladder to tell whether the condition is benign or malignant, because true malignancy may depend upon the disintegration of the base of the tumor, that is, whether or not the basement membrane has been broken through and the underlying tissues infiltrated by the proliferating epithelial degeneration.

TRANSACTIONS

OF THE

NEW YORK SURGICAL SOCIETY.

*Stated meeting, held at the New York Academy of Medicine,
November 12, 1913.*

The President, DR. FREDERIC KAMMERER, in the Chair.

FRACTURE OF THE SCAPULA.

DR. JAMES M. HITZROT presented two young men, each of whom had sustained a fracture of the scapula, one in attempting to stop a runaway horse, the other in a fall from a truck. In the first case, the X-ray showed a fracture through the neck of the scapula. The characteristic deformity usually described in connection with this form of fracture was absent, and there was practically no loss of function. No effort at reduction had been made.

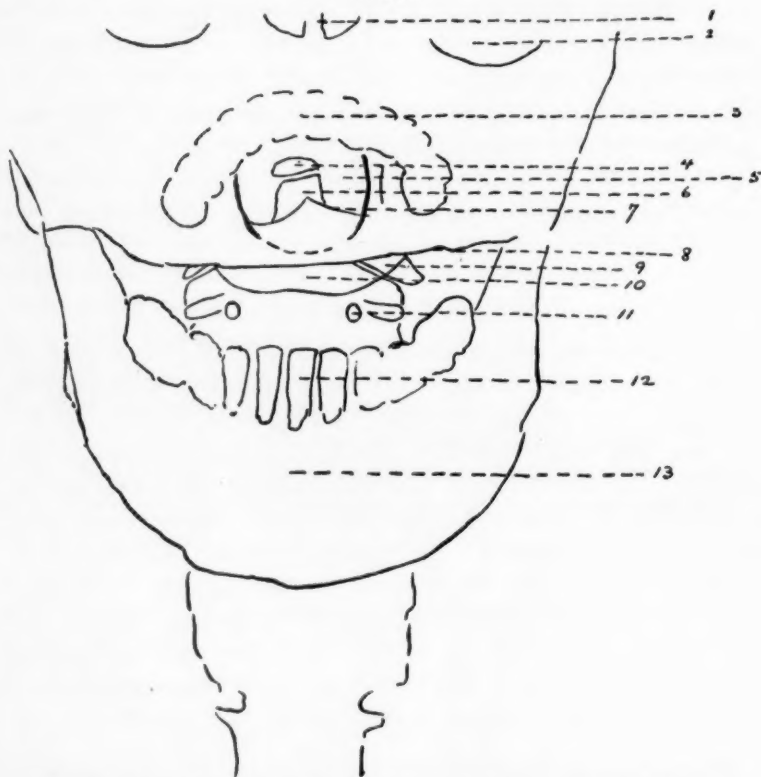
In the second case, the fracture was through the lower facet of the bone, and the lower two-thirds of the articular surface had been carried downward and inward, producing loss of function. The clinical features of this case were fairly characteristic, and there was a marked difference between the two shoulders.

FRACTURE OF THE ODONTOID PROCESS OF THE AXIS.

DR. OTTO G. T. KILIANI presented a man (and the X-ray plate) who was riding to hounds eight weeks ago, when his horse, a heavy English hunter, stepped into a deep, narrow ditch, and in endeavoring to recover itself, threw its head back, striking the rider in the face and breaking his nose. He was stunned, and pitched forward, falling to the ground upon his face. A short period of unconsciousness followed, no vomiting; after two days' rest, he attended to his usual duties. When Dr. Kiliani saw him, about ten days later, the only symptom he complained of was

inability to raise his head while lying down. No pain on rotation, some pain on flexion to either side, and on flexion backward. An X-ray was taken through the open mouth, which clearly revealed a fracture of the odontoid process of the axis, the top of the process being broken off.

FIG. 1.



Tracing of X-ray plate taken by Dr. W. H. Stewart, visiting radiologist of the German Hospital. Photograph taken with the mouth open, the patient in a recumbent position, with the occiput on the plate. 1, anterior nasal spine; 2, orbit; 3, upper teeth; 4, fragment; 5, foramen magnum; 6, odontoid process; 7, internal occipital crest; 8, lower line of base of skull; 9, transverse process of atlas; 10, anterior arch of atlas; 11, intervertebral foramen; 12, lower teeth; 13, lower jaw.

Dr. Kiliani said that a rather careful search of the literature showed that this form of fracture had either gone unrecognized, or was extremely rare. In most of the cases reported, the fracture was through the neck of the process, lower down than in this case. Dislocations of the axis, of course, were comparatively frequent. As to the explanation of the fracture in this instance,

one could only surmise that it was possibly the result of a sudden, forcible flexion of the head backward, or that the fragment had been torn off by its ligamentous attachments.

EXPERIMENTAL INTESTINAL OBSTRUCTION.

DR. JOHN A. HARTWELL presented a dog to illustrate the results of some experimental work that had been done by Dr. J. P. Hoguet and himself bearing upon the subject of intestinal obstruction, the object being to find out the direct cause of death in this condition. The dog had been operated upon twelve days previously, when the bowel was sectioned at the lower end of the duodenum and the two ends closed by inversion, thus producing a complete obstruction. The amount of urine and vomitus put out was measured, and the dog received, subcutaneously, normal saline solution in amount slightly in excess of this. During the first four days of the experiment the output was approximately 450 c.c. per day, which was exactly one-twentieth of the body weight. After the sixth day the vomiting ceased and the stomach tube recovered nothing. The amount of urine, however, increased, and corresponded closely to the amount of saline given. The animal was in perfect condition except for loss of weight, due to lack of food.

Dr. Hartwell stated that in this way they had been able to keep dogs in a healthful condition as long as twenty-six days, which was the longest they had continued their observation, but under this treatment no animal in a series of ten had died. On killing these animals, examination of the organs showed a normal liver, kidney and spleen, the only lesion found being a dilatation of the intestine and stomach above the obstruction. Dogs so operated, who did not receive the salt solution, lived never more than ten days, and usually died in five or six. Autopsy in these cases showed a very marked granular degeneration of the kidney and liver, with sometimes actual necrosis of the latter. A comparison of the urine in the treated and untreated dogs showed that of the former to be absolutely normal, while that of the latter contained albumin in excess, two to three times the normal output of nitrogen, and a marked disturbance in the creatin creatinin ratio. These findings demonstrate conclusively a very marked destruction of tissues, which is accounted for by the abstraction of water. Dogs with a low intestinal obstruction behave in exactly

the same way, with one important difference. In case the over-distention of the bowel above the obstruction becomes so great that the circulation is seriously disturbed, with a resulting damage to the mucosa either in the way of ulceration or necrosis, the dogs cannot be kept alive by the administration of the salt solution.

Dr. Hartwell said that further experimentation they had done confirmed this finding; the damage to the mucosa introduced a new factor into the situation, and that when this was present, saline solution would not save life. The experiment, therefore, demonstrated that the cause of death depends upon two factors. First, the loss of water from the tissues, and second, the damage to the intestinal mucosa, from which, apparently, poisons were absorbed, this being the only source of actual poisoning. Though no direct evidence was obtained, microscopical examination of the damaged mucosa indicated that the poison arose from an infectious process in the damaged mucosa.

Dr. Hartwell said that a clinical application of these findings had been made in two cases of intestinal obstruction, who had absorbed 7000 c.c. of salt solution, administered subcutaneously, during the first thirty-six hours following the operation.

Further, it would seem advisable to empty the bowel above the obstructed point when the obstruction had persisted long enough to result in damage to the mucosa, and if the damage was extensive, an enterostomy probably would be of benefit in order to maintain drainage for a few days.

JEJUNAL ULCER FOLLOWING GASTRO-ENTEROSTOMY FOR DUODENAL ULCER.

DR. HARTWELL presented a man, thirty-three years old, who was admitted to Bellevue Hospital in June, 1907, with the following history: He was a truckman, working very hard, and drinking both beer and whiskey to excess. There was no record of any previous illness. His present illness dated back about one year, during which time he had three attacks of acute abdominal pain in the right lower quadrant of the abdomen. The suddenness and severity of these attacks is witnessed by the fact that he has been seized with them when at work or walking, and has been suddenly incapacitated by the intense cramp-like pain in this region. He has usually vomited at the time of the onset. The pain had lasted severely only for a short time, but he has re-

mained in bed from two to ten days. He was just recovering from an attack on his admission to the hospital. Examination that time showed slight tenderness and rigidity in the appendix region; nothing else. There was no elevation in temperature. An appendectomy was done, and a slightly inflamed appendix was removed.

Following this operation he was entirely free from symptoms until November, 1909, when he had another very acute attack of abdominal pain which made him fall to the ground. It was accompanied by vomiting and great prostration. He was immediately taken to Bellevue Hospital, where an operation was done for an acute perforation of a pyloric ulcer. The ulcer, which was on the anterior wall, was closed with purse-string suture, and a posterior gastro-enterostomy with short loop by means of the Murphy button was done. His recovery from this operation was completely satisfactory and he remained in good health until May, 1913. He then again began to suffer moderate abdominal pain, this time mostly in the epigastrium, sometimes accompanied by vomiting. The pain usually began in the morning, shortly after breakfast, and continued throughout the day, irrespective of meals, but relieved by vomiting. Blood was noticed in the vomitus only once, and there was no record of his having passed any by stool. After three or four weeks of this condition he came to the hospital and was operated upon by another surgeon. The gastric artery, with the nerves surrounding it, was doubly ligated; the object being to modify the secretion of hydrochloric acid. Some relief was obtained following this operation, but in a few months the symptoms recurred exactly as before. He was readmitted to the hospital early in October, 1913. Examination at that time showed him to be in good condition, well nourished, and suffering only from a recurrence of his previous symptoms. There was a ventral hernia at the site of the previous operations. No points of tenderness were present, and no masses could be felt. A bismuth X-ray showed that the stomach emptied itself rather promptly and that both the pylorus and the gastro-enterostomy were open. Gastric analysis after a test meal showed a total acidity of 60; combined acidity 48; free HCl 40. No lactic acid; no blood; many starch granules and undigested food particles. The aspirated material was bright green in color, which was found to be due to the presence of bile pigments.

Operation was performed by Dr. Hartwell on October 10, 1913, through a median incision in the epigastrium. On opening the peritoneum, rather extensive adhesions were found between the great omentum, the pylorus and the gastro-enterostomy stoma. On freeing these adhesions, the scar of the old ulcer on the pylorus close to the gastric side of the pyloric vein was palpable. The pylorus readily admitted the index finger on invaginating the stomach through it. The stomach itself was moderately dilated. The gastro-enterostomy opening easily admitted two fingers. The jejunum on its anterior surface, immediately distal to the anastomosis, was adherent to the wall of the stomach about one and one-half inches from the line of union. On separating the adhesion, which was comparatively recent, a perforation was found in the jejunum about three-eighths of an inch from the anastomosis. The ulcer, of which the perforation was the centre, was excised, together with the line of anastomosis adjacent to it. The opening into the two viscera thus made was closed by the usual suture method, re-establishing the stoma. Convalescence following this operation was satisfactory, and the patient has been entirely free from any symptoms. His diet, which has been somewhat limited, has been taken without discomfort or nausea.

A gastric analysis, made on November 5, shows a total acidity of 50; combined acidity, 15; free hydrochloric acid, 12. No lactic acid; no blood nor bile.

Dr. Hartwell said this case was presented to emphasize several facts: First, that the patient's original symptoms were probably due to the pyloric ulcer, but were relieved by the appendectomy. Unfortunately, no gastric analyses were made at that time. His entire freedom from pain from this time up to the acute perforation two years later is unusual considering the fact that he had previously suffered from attacks of severe pain. The fact that bile was found in considerable quantities in his gastric contents before the last operation is worthy of note, in view of the belief that these marginal ulcers occurring around the gastro-enterostomy stoma are in part caused by the passage of unneutralized hydrochloric acid over a mucosa that normally is not subject to this irritation. The fact that so many of these ulcers occur at or near the stoma makes it seem probable that the operative trauma is an etiological factor. For this reason, the writer has of late discarded the use of the clamps in this operation, and

followed the method of Coffey, in which the soiling of the abdomen is prevented by elevation of the stomach and jejunum by the use of traction sutures.

The case is also presented as one more illustration of the fact which we all recognize, namely, that surgical means are at best but an aid toward the cure of gastric and duodenal ulcers. This patient has admittedly been as careless in his diet and in the use of alcohol during the past three years as he was previous to his original trouble. This factor probably has an important bearing on the recurrence of his symptoms.

DR. ROBERT T. MORRIS said that in one case where he excised a jejunal ulcer there was a recurrence about a year later. The patient died from inanition, and at the autopsy a second ulcer was found about two inches below the primary one. If these ulcers were due to the highly acid character of the stomach contents bathing the newly exposed mucosa, we may fairly assume that its irritating effect is exerted in causing proliferating endarteritis over the areas of distribution of some of the terminal arteries, resulting in a localized anæmia and the formation of ulcer. When this process was under way, it seemed to call out a general or local leucocytosis which acted as a protection against further involvement of the other terminal arteries in the vicinity. The above, at least, Dr. Morris said, was a working hypothesis.

ARTERIOVENOUS ANASTOMOSIS FOR THROMBO-ANGELITIS OBLITERANS.

DR. HOWARD LILIENTHAL presented a man, forty-four years old, married, a Russian by birth, who was admitted to the Mt. Sinai Hospital on March 1, 1913, with the history that his illness began eighteen months prior to that time with the appearance of painful red streaks on the legs and knees, the discoloration migrating down to the feet. Eight weeks before his admission, painful swelling of the right foot developed, and three weeks later a physician had incised the right great toe without evacuating any pus and without relief from the pain. The wound failed to heal and had become gangrenous and excruciatingly painful.

On admission, there was no pulsation in either foot, and both feet and legs were mottled in appearance. There was frank gangrene of the right great toe.

After the disease and its prognosis were explained to the

patient, together with the possible necessity for amputation, he consented to an arteriovenous anastomosis, which Dr. Lilienthal performed on March 10, 1913. Through an incision from the groin to the mid-thigh he made the anastomosis in Hunter's canal, employing the method of Carrel, closing off by ligature the continuation of the artery below and of the vein above. In the event of failure it was believed that some circulation might be carried on through the vessels given off above the arterial section.

As soon as the suture was completed and the clamps removed, the vein filled, and a small tributary just below the level of the union spurted arterial blood and had to be ligated. The only change in the foot was the appearance of a patch of pallor about the gangrenous area. The wound was closed with deep catgut sutures and a superficial layer of silk. On the following day the patient's temperature rose to 101° F., but there was no change in the appearance of the foot. On the second day, however, it had a better color than its fellow, but there was no relief of the pain and no venous pulsation had appeared. A month later there was great improvement, all the oedema having subsided and the skin had resumed its natural color.

In spite of a negative Wassermann reaction, salvarsan had been administered, but the pain in the toe was so severe and persistent that it was amputated on April 11. At this operation there was considerable bleeding from spurting vessels, something which the speaker said he had never before observed during an amputation for this condition.

Since the patient's discharge from the hospital, about six months later, there had been gradual but certain improvement. The wound in the foot healed slowly, and even at the present time there was a minute spot where the skin previously healed had been rubbed off. The general appearance of the foot, however, was quite normal, and there was no more pain, which he regarded as one of the most important criteria of the success of the operation.

DR. LILIENTHAL presented a second case of arteriovenous anastomosis for thrombo-angeitis obliterans, in the person of a man, fifty-four years old, who was admitted to the Mt. Sinai Hospital on March 22, 1913, with the story that four months before that date he had applied a solution of carbolic acid to his left foot, and that as a result there had appeared a spot of gangrene

upon one toe. On admission, he complained of pain principally in the middle toe of the left foot, and there was here a darkening of the skin, with some necrosis of the soft parts. Neither popliteal artery could be felt, and there was no dorsalis pedis pulsation in either foot. The middle toe of the left foot showed skin necrosis extending from the nail for a distance of one and a half centimetres upon the plantar surface. This region was extremely tender to the touch and very painful.

On admission, the patient's temperature was 99.2° ; pulse, 92; respirations, 28. His general condition was good. The left foot, in addition to the lesion on the middle toe, was slightly dusky, and there was a trace of œdema upon the dorsum. The Wassermann blood test was negative.

On March 24, 1913, under ether, an incision was made from the highest point over the femoral vessel down to the level of the lower portion of Hunter's canal. The vessels were carefully dissected out, and a temporary ligature was placed upon the vein at a point about one centimetre from its entrance to Hunter's canal. This ligature was merely crossed and held with an artery forceps, and was placed in such a way that it embraced a large tributary, as well as the vein itself. The upper portion of the vein was then permanently ligated and a section made below the ligature, a considerable distance above a large valve. The blood was washed out of the lumen of the vessel with Ringer's solution. The femoral artery was now freed, and a ligature passed around it as far below the section of the vein as possible. Just before this ligature was tightened, however, the forceps holding the femoral vein ligature slipped, and there was a sudden and most annoying hemorrhage. Although the actual loss of blood was not great, the conditions were rendered so unpromising that he was almost induced to abandon the operation and simply ligate the vein. However, the vessel having been secured with the fingers, and the large tributary having been permanently ligated, it was decided to proceed with the operation. A serrafine now took the place of the ligature, the artery was permanently ligated, and while it was controlled from above with two serrafines, section was made just above the ligature. There was considerable slack owing to the extra length of the artery, but thinking that the operation would thus be rendered easier, this slack was not cut away. An arteriovenous anastomosis was now completed by Carrel's method, using No. 13

needles and thread six O. The anastomosis was rendered somewhat difficult because of the great disparity in size between the small femoral artery and the unusually large femoral vein. When the suture was completed and the clamps removed, the vein immediately filled out, and on manipulating the vessel there was a spurt of blood, showing a leak at the line of suture. Two extra sutures were inserted and the clamps were again removed. There was now no leakage, but on moving the vein and artery so as to angulate the line of anastomosis, blood again showed. This, however, stopped spontaneously, nor could it again be made to flow without the use of undue and dangerous manipulation. Feeling a bit uncertain, however, as to the exact condition of the anastomosis, a ligature of chromicized catgut was passed around the femoral vein and another loosely about the femoral artery, and the wound was then closed completely, without drainage, the long ends of the chromicized gut being buried by the cutaneous suture. This was done so that in case of accidental hemorrhage, the house surgeon might easily remove the skin sutures and find the ends of the chromicized gut ligatures ready for tightening. The necessity for this, fortunately, did not arise.

Immediately after the operation the affected foot looked slightly more cyanotic than its fellow. Eighteen hours later the color of the foot was excellent, with an apparently good capillary circulation. The patient had suffered no shock nor severe pain.

Dr. Lilienthal said this was the second case of localized gangrene of the foot in which the condition was ascribed to the use of a carbolic acid lotion. Personally, he did not believe that the use of the carbolic acid had anything to do with it. The speaker said he was naturally interested in the outcome of this operation, as he was the first one to perform it in this way, although he had been anticipated by Hubbard who used another operative method. The exact relation of the operation to the cure was very questionable, in his mind. He had been informed that in these cases relief would follow simple ligation of the vein, but he preferred to do an arteriovenous anastomosis, in spite of the fact that the results were not very convincing to him, as he had never been able to get any venous pulsation excepting on the operating table. Still, the fact that he had been compelled to tie spurting vessels after this operation indicated an arterial circulation, and he

could not recall any case of amputation for gangrene resulting from thrombo-angeitis obliterans in which he had found it necessary to ligate a vessel unless the amputation was done very high up—certainly never before during an amputation of the toe, as in the first case shown. Whether the benefit derived by these patients resulted from an obliteration of the vein by thrombosis, or whether there was an actually functioning arteriovenous anastomosis he did not know. He felt assured, however, that both of these patients would have been subjected to an amputation of the foot but for this operation.

DR. C. A. McWILLIAMS said that at one of the meetings of this Society last spring he showed a patient upon whom he had done an anastomosis between the femoral artery and vein by the Bernheim method, which he thought was preferable to the end-to-end method because there was less liability of subsequent interference with the arterial circulation. In that case, antedating the operation, the patient had suffered from a gangrenous toe, and after some hesitation, Dr. McWilliams said he amputated the toe and the wound healed very nicely, with the exception of a small sinus. The patient was markedly relieved of his pain.

One added advantage of such an anastomosis in this condition, the speaker said was that in case amputation subsequently became imperative it could be done lower down than otherwise.

DR. EDWARD M. FOOTE said that in his experience with these cases, the pain had varied greatly at different times. In one case in which during a period of eight years he had found it necessary to amputate fragments from all four extremities the pain had been very severe at times, while at other times it was comparatively trifling. As some of these patients are worse in the winter, he had rather held the idea that cold weather was an important factor in the causation of this condition; but recently he had a patient from San Domingo with typical well advanced lesions.

DR. LILIENTHAL, in closing, said he had never seen any satisfactory explanation for this condition. He was not inclined to attribute it to cold weather, nor would that explain why it was practically confined to men, and to young men as a rule. In all of his cases, the patients were addicted to the excessive use of cigarettes.

The speaker said he had seldom found a thigh amputation necessary in these cases. He usually amputated according to the

condition of the vessels, and he would not go as high as the thigh unless it was absolutely indicated. He had repeatedly used Dr. Moschcowitz's modification of Bier's method and done an osteoplastic amputation, with very good bone nutrition. An operation in these cases should not be looked upon as a cure, but simply as a relief measure. There was no cure for these patients.

As to the matter of ligating the vein, some authority—Coenen, he believed—claimed that relief was obtained in that way by keeping more blood in the affected parts. However, after the production of Bier's hyperæmia in these cases, some of the patients complained bitterly of pain. As to the side-to-side method of anastomosis, which Dr. McWilliams preferred, the speaker said he did not approve of it unless we admitted that we were simply accomplishing a ligation of the vein, because he was fairly certain that with this method of anastomosis there was a strong tendency to the production of a saddle thrombosis.

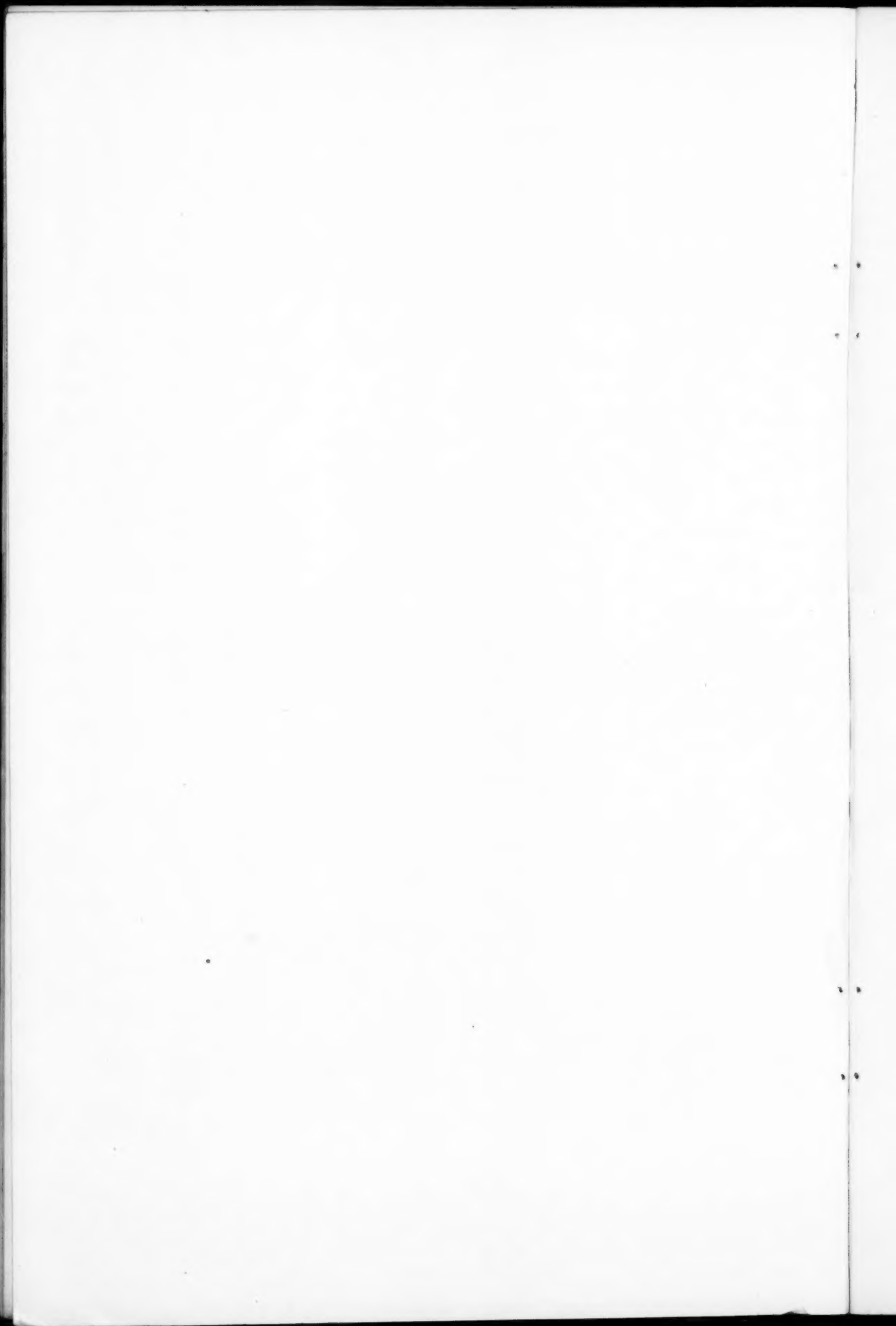
MIXED TUMOR OF THE PAROTID GLAND, WITH MALIGNANT DEGENERATION.

The President, DR. F. KAMMERER, showed a woman of forty-eight years upon whom he had operated for a mixed tumor of the parotid gland, with malignant degeneration, in July, 1913. The patient had noticed a slowly growing tumor below the left ear for the past 3 or 4 years. During the past six months it had increased very rapidly in size, until, at the time of her admission to the hospital, it presented the appearance shown in the accompanying photograph. (Fig. 2.) An elliptical incision was made over the tumor and extended downward to expose the external carotid artery, which was ligated with some difficulty. The extirpation of the growth was accomplished without much hemorrhage. The tumor had entirely eroded the zygomatic arch. The ramus of the lower jaw near the angle had to be removed, with the exception of a small ridge which preserved the continuity of the bone. The buccal cavity was not opened. There was apparently no involvement of any of the lymphatic glands. The facial nerve was naturally divided during the operation. The patient made an uninterrupted recovery, and up to the present time there had been no recurrence. The microscopic diagnosis was carcinoma.

FIG. 2.



Mixed tumor of the parotid gland. (Kammerer.)



The speaker referred to a similar case of carcinomatous degeneration of a mixed tumor of the parotid, not quite as large as the one shown this evening, which he had operated on four years ago. In that case there was no local recurrence, but the patient died 3½ years later of what seemed to have been metastatic deposits in the liver.

In connection with these two cases, Dr. Kammerer had hoped to show a third case of a large parotid tumor, which he had operated on eight years ago, a young woman who was in perfect health at present. In this case, although an operation was done for a second recurrence, the growth showed no malignant degeneration.

In mixed tumors of the parotid or submaxillary gland, which have very gradually increased in size, it was certainly the proper surgical procedure to enucleate them from the investing capsule, leaving the salivary gland intact. For such cases simple enucleation should lead to a permanent cure. The speaker, however, thought that after one or two recurrences it was advisable to do a radical operation, removing the entire gland, even at the cost of the facial nerve, in tumors of the parotid, more especially as statistics seemed to show that the repeated traumatism of operative interference was a decided factor in the conversion of mixed tumors to the malignant type.

ABSCESS OF THE LUNG: INCISION AND DRAINAGE: THORACOTOMY.

DR. LILIENTHAL presented a boy, thirteen years old, who was admitted to the Childrens' Service of Mt. Sinai Hospital late in December, 1912, where for some time he was under the observation of Dr. Henry Koplik. The history obtained was that he had a left-sided pneumonia in infancy and that his tonsils and adenoids had been removed a year and a half ago. It was said that he had a second attack of pneumonia a few months ago, and following this he suffered from chronic cough and an occasional hæmoptysis. About two weeks before his admission his cough became worse; he was feverish, complained of pain in the chest and was easily tired.

A physical examination showed dulness over the left supra-

spinous fossa extending to one finger below the spine of the scapula. There was also marked dulness anteriorly over the left side and the upper part of the left axilla, with bronchial voice and breathing over these areas. There were some subcrepitant râles over the left supraspinous region, with bronchovesicular breathing over the left interscapular region. The breathing sounds were much diminished over the left base, together with impaired resonance. A blood count showed 15,000 white cells, with 54 per cent. of polynuclears. The von Pirquet test was negative, and no tubercle bacilli were found in the sputum.

On December 21, 1912, pus was obtained on aspirating in the left axilla, the needle being carried upward and backward. About two weeks later the boy was anesthetized, and an incision was made from the clavicle downward, dividing the fibres of the pectoralis major, which were then bluntly separated and the second rib exposed. A section of this rib, about three-quarters of an inch long, was removed, and upon aspiration, foul pus was again withdrawn. Upon enlarging this opening with a grooved director and dressing forceps, it was noted that in passing through the abscess cavity the tissues were extremely hard—almost cartilaginous. About three drachms of thick foul pus were evacuated. A tube was then inserted and the wound packed. Prior to this operation, the X-ray had shown a diffuse opacity of the left chest, but nothing like a lung abscess could be made out. At the time of the operation, beneath the thick-walled abscess, apparently normal pleura could be seen moving with respiration. The general pleural cavity, however, was not entered, as the case was apparently one of lung abscess connecting with the bronchus. Dr. Koplik did not agree with this diagnosis, regarding the case as one of apical empyema.

On January 7, 1913, the tube was removed, a strip of tape impregnated with bismuth was packed into the wound, and a stereoscopic X-ray picture taken in the hope of demonstrating the exact location of the lung abscess. Owing to the rather diffuse area of opacity of this entire region of the chest, however, the subsequent picture proved little excepting that the cavity was nearer the posterior than the anterior wall of the chest. The case progressed favorably for about ten days, when the patient again became feverish and complained of considerable pain in the wound. Under nitrous oxide anesthesia the drainage

opening was stretched so as to permit of digital exploration. Nothing of note was found, and aspiration practised in various directions upward gave negative results. There was still considerable cough and expectoration.

On January 25 the wound in the chest wall had filled up and showed a strong tendency to heal. The cough and expectoration, however, persisted, with occasional rises of temperature. Flatness in the upper, posterior part of the chest extending to the axillary region caused Dr. Koplik to suspect the presence of fluid, probably pus, and under a general anæsthetic the chest wall was again aspirated, the needle being passed through the upper portion of the axilla, pointing upward and backward. Although a large sized needle was used, no pus was obtained at this puncture. At one attempt, pure blood was withdrawn, perhaps a drachm, before the suction was checked. The anterior wound was now explored with the finger, but only the original cavity was found. A needle inserted here and passed through the posterior wall of this cavity withdrew a few drops of extremely thick pus. A dressing forceps following the needle was pushed through until the points were palpable underneath the skin just above the scapula. Here a counter incision was made and a drainage tube of considerable size was drawn through from the back to the front.

Following this operation, the boy's condition gradually improved. The fever and the quantity of the expectoration varied from day to day, but gradually diminished until the drainage tube was removed, the sinus being kept open by a large-sized triple silk ligature. When this was withdrawn, on March 5, the patient still had a pallid, sick appearance, although his nutrition had improved. Upon the withdrawal of the silk, the cavity was injected with iodoform sesame oil and spermaceti (Mosetig-Moorhof filling), and for a long time after this was done the patient complained of the taste of the iodoform. The wound was now nearly healed and the general improvement continued, though cough was still present.

The patient left the hospital on April 25, 1913, and was subsequently sent to a convalescent home, where he remained for some time and his health steadily improved. His condition at the present time was excellent.

POST-OPERATIVE INTESTINAL OBSTRUCTION: ENTERO-ENTEROSTOMY.

DR. EUGENE H. POOL presented a man, twenty-four years old, who was admitted to the New York Hospital on July 8, 1913, with the history that for three weeks prior to his admission he had been confined to bed with an attack of appendicitis. Examination revealed a tender mass, about 2 x 3 inches, in the right lower quadrant. The patient was operated on and an appendiceal abscess was opened and drained. The patient did well until the fourteenth day after the operation, when he complained of pain in the abdomen, with frequent retching and vomiting, and later hiccoughs. He was given a high enema, which was fairly effectual, but his symptoms continued throughout the night and the following day. Upon washing the stomach, which was done three times, a greenish fluid was recovered. Repeated enemata and colon irrigations at first brought away a small amount of gas, finally neither gas nor feces. On the morning of the sixteenth day after operation (48 hours after the onset of the symptoms) the patient was vomiting at frequent intervals small amounts of dark-colored fluid, and was hiccoughing. There was absolute obstipation. While there was no general abdominal distention, visible peristalsis was noted in the upper abdomen.

The abdomen was opened to the right of the midline above the umbilicus; the right upper quadrant and appendical regions presented a mass of firmly adherent intestines. A greatly distended loop of intestine was visible; likewise collapsed loops of small intestine. Three distinct deep-lying bands of adhesions were freed, but in spite of this the bowel remained collapsed, and as it seemed unwise to further prolong the search, a lateral anastomosis with suture was made between the collapsed and distended loops close to the adherent mass. The patient vomited only once after the operation and his immediate recovery was uneventful. Fourteen days after the anastomosis, however, he again complained of pain in the abdomen and vomited a large amount of fluid. The symptoms then temporarily subsided, but five days later they recurred with increasing severity, with every evidence of complete high intestinal obstruction. The abdomen was again opened, this time to the left of the midline above the umbilicus, revealing a condition similar to that found at the preceding operation, excepting that the adhesions were even

more extensive. The previous anastomosis was found surrounded by adhesions. There was a short section of greatly distended bowel, showing that the obstruction was high in the small intestine; a second anastomosis was made between this and the collapsed bowel, a short distance from the adherent mass. The wound was closed without drainage, and the patient made a good recovery. With the exception of occasional attacks of colicky pain, he was now in good health and his bowels moved regularly without the aid of cathartics, upon which he had to rely before entering the hospital.

Dr. Pool said the case was shown to emphasize the advisability of doing an anastomosis in cases of this kind. Where the obstruction is very high in the intestinal tract, an enterostomy is to be avoided; therefore, in the presence of multiple bands and adhesions where the obstructing band cannot be readily found or efficiently dealt with an entero-enterostomy may be performed to advantage.

THE DIETETIC TREATMENT OF GANGRENE IN DIABETES MELLITUS.

DR. A. V. S. LAMBERT read a paper with the above title, for which see page 176.

DR. MORRIS thought it very important to consider the character of these infections. Sugar in the blood exerted a hygroscopic action, and extracted water from the normal cells thereby lessening their efficiency as phagocytes or as repair cells. The speaker said he felt convinced that these patients did very much better from a surgical point of view if they were placed on a standard diabetic diet and one that prevented intestinal putrefaction. Dr. Morris said he had brought with him a radiograph demonstrating the calcification of the arteries that Dr. Lambert had described and also a dietary list similar to the one described by Dr. Lambert.

DR. HARTWELL said that at Bellevue Hospital, where they had many of these cases, most of them were treated in the medical wards and received appropriate diet under the supervision of Dr. Graham Lusk. Here the dietetic treatment was much more rigorously carried out than it could be in a general surgical ward. The results had been more satisfactory and much better than when one relied only on the usual surgical methods.

APPARATUS FOR RECTAL ANÆSTHESIA.

DR. EDWARD M. FOOTE described this method, which had recently been first successfully employed by Dr. James T. Gwathmey. It consisted, essentially, of the administration, per rectum, of a 60 per cent. mixture of ether in mineral oil. A cathartic and enema were given the day before operation; while about an hour before operation a hypodermic of morphine was administered. Then the mixture of ether and oil, slightly warmed, was injected through a rectal tube by means of a funnel. The introduction was made slowly, usually employing about four ounces of ether and two ounces of oil. An ounce of ether by measure to thirty pounds of body weight was a safe dose to use. The ether was absorbed gradually but rapidly so that the smell of ether could be detected in the breath in about five minutes, and within fifteen minutes the patient fell quietly asleep, without any struggle or muscular excitement. The patient was under the impression that he was receiving an ordinary enema, and thus mental shock was eliminated, there was no strain on the heart, no interference with respiration, no coughing nor swelling of the mucous membranes. Dr. Gwathmey had now employed the method in over 50 cases. He employed a mixture composed of 75 parts of ether to 25 of olive oil. After the patient was thoroughly under its influence, the excess should be washed out of the bowel. It apparently caused no irritation of the rectum nor of the kidneys, and was eliminated largely by the lungs.

Dr. Foote said that personally he had tried the method in thirteen cases, and so far as his experience with it went, he regarded it as one of the greatest advances in our knowledge of anæsthesia. In some cases where the peritoneum, or other sensitive parts had to be handled, he had found it necessary to give a few drops of ether by the ordinary method. In about one-half of his cases, there was some nausea, but decidedly less than by the ordinary method of etherization. In trifling operations, where prolonged etherization was not necessary, it was not indicated: there nitrous oxide was preferable.

DR. POOL said he had tried this method of etherization in one case at the French Hospital. The operation was a comparatively slight one. The details of the anæsthesia were as Dr. Foote described, but the patient remained in the same condition for four hours.

BOOK REVIEWS

ANATOMY, Descriptive and Applied, by Henry Gray, F.R.S.A., new American from the eighteenth English edition, thoroughly revised and re-edited. By ROBERT HOWDEN, A.M., M.B., C.M., Professor of Anatomy in the University of Durham, England. Philadelphia and New York: Lea & Febiger, 1913.

The nineteenth edition of Gray's Anatomy is a decided improvement over many of the previous editions. The editor has carefully gone over the various chapters, making many needed corrections and additions. Many of the original figures have been removed and new ones added together with a considerable rearrangement of the subject matter.

One of the most important improvements in this volume is the adoption of the Basle nomenclature. The need of this change alone warranted a new edition and the change will be welcomed certainly by the students using the text. Prof. Howden has judiciously used many of the Latin terms directly and in other cases given literal translations of these terms. In but few instances have the old names been used for the translation; for example, the name sciatic has been substituted in the place of the old name, great sciatic, or the Basle name ischiadicus. In other cases, names like anterior nasal aperture, the editor has justly retained in the place of the Latin name, apertura piriformis.

The value of some of the rearrangements in the text may be questioned. Undoubtedly a wise change was made by collecting together into one chapter at the end of the book the various parts given over to surface anatomy. Surface anatomy forms a separate branch of the subject and is best discussed subsequent to a descriptive study of the various parts of the body.

The collecting together of the various parts given over to histology and embryology into one chapter at the beginning of the book are perhaps of much less importance. It is true all text books of anatomy should contain a chapter dealing with fertiliza-

tion, segmentation, and the development of the primary germ layers. The later and more detailed discussion of the development of the organ may be more fitly placed, however, in the chapters dealing with a description of these parts, serving here to make clear the relation and the significance of the various structures. This is especially true in the descriptions of the gastro-intestinal tract, the genito-urinary system, as well as several other organs and tissues.

The publishers have likewise shown a decided interest for the improvement of this volume. The substitution for the harsh and highly colored illustrations of blood-vessels of ones tinted with paler blues and reds and showing some of the general contour of the vessels is much welcomed.

A general criticism that may be justly made of this as well as former editions of the book is the narrow impression of the scope of the subject conveyed through the parts on applied anatomy. The parts deal largely with surgical applications of anatomy. They are useless to the beginning student untrained in clinical branches and still useless on account of their brevity for the student of surgery. Anatomy forms the foundation of all clinical diagnosis and unless it is presented so as to convey to the student its relation to the solution of problems dealing with the life of the individual it remains no longer a science but becomes a mere memory drill.

Aside from this general criticism the reviewing of this book has been a pleasure, since it undoubtedly represents the beginning toward a return of Gray's Anatomy to the high standard it once enjoyed as a text-book of descriptive and applied anatomy.

M. T. BURROWS.

DISEASES OF THE STOMACH. BY GEORGE ROE LOCKWOOD, Professor of Clinical Medicine, Columbia University; Attending Physician, Bellevue Hospital. Octavo, 624 pp. Lea & Febiger, Philadelphia and New York, 1913.

The work is based on the personal observations of the author from material drawn both from private and hospital sources, and represents essentially his individual opinions on diseases of the stomach, his large experience offering him exceptional oppor-

tunities in this line of work. He has, therefore, attempted to state statistics as he has compiled them without regard to previous views held by other authors. This novel method of presentation is plainly noticeable throughout the entire text and it is particularly pleasing to note the absence of the usual historical items, both in text and illustrations, which are so frequently placed for some unknown reason in every text-book.

The text is divided into twenty-one chapters, each practically a monographic report of Dr. Lockwood's own observations, and cover all the essential pathologic conditions present in the stomach. The consideration of surgical intervention in various conditions is fairly stated and judicious in its scope. The freedom of quotation of foreign surgeons and clinicians and the relative paucity of reference to American observers may be noted, as certainly the best work in this field is at present being done here.

The propriety of introducing text and illustrations of methods discarded even by the author himself, as in the case of intragastric faradism, page 330, may be questioned, as may also be the retention of such terms as heart burn, page 478, as a heading which is not only inaccurate but misleading and incorrect.

The dietetic and medicinal treatment of the various disorders is exceptionally commendable and marks a definite departure from previous works in that one does not continually remark that indefinite generality usually accorded these important subjects. A careful consideration of both these methods of treatment, as described by Dr. Lockwood, cannot but be of the greatest benefit not only to the internist, but also to the surgeon.

Differences of opinion may be formed naturally to many statements made, as for instance, that 60 per cent. of patients over fifty years of age have achylia, or that there is a thorough digestion of bread stuffs in the stomach (page 535). But the text is so replete with accurate and scientific observation that these minor differences may be overlooked. Typographical errors, such as the word secretion instead of secretin (pages 509 and 329) will undoubtedly be corrected subsequently.

The introduction of charts to represent the time occurrence of pain is a particularly noteworthy innovation and saves much descriptive text. The illustrations, particularly the radiographic plates, are excellent owing to skilful and commendable retouching.

The book is one which will certainly repay the reader, and

is deserving of close study at this time particularly, when we are gradually entering into phases and treatment of diseases evolved from the physiology of internal secretions and fundamentally based on the pathology of the living rather than the dead.

JAMES T. PILCHER.

CARBON DIOXIDE SNOW. Its Therapeutic Uses. By J. HALL-EDWARDS. Birmingham, London: Simpkin, Marshall, Hamilton, Kent & Co., Ltd. 1913.

In this little volume the author sets forth the methods of collecting, preparing, transporting, and applying carbon dioxide snow in a clear and readable manner. In addition to giving the ordinary means of collecting the snow, the writer describes the use of special apparatus which he has designed for the purpose. The manner of application, effects, length of exposure, and amount of pressure necessary are gone into very thoroughly, and while reference to cases treated has been purposely omitted, a list of diseases in which the snow may be successfully used is given, and the treatment of those conditions in which snow has been most extensively employed is described in detail. The printing is excellent; there are numerous illustrations, and the book may be commended as a reliable guide to those desirous of trying this form of treatment.

JOHN A. C. MACEWEN.

CORRESPONDENCE

THROMBOSIS OF THE MESENTERIC VESSELS.

EDITOR ANNALS OF SURGERY:

Inasmuch as thrombosis of the mesenteric vessels is so infrequently seen, so rarely diagnosed preceding exploratory laparotomy and post-mortem examination, and knowing that the mortality in this disease is so extremely high, 92 per cent. to 94 per cent. (Jackson), I thought it would be of interest to some one to report a case which was referred to me for operation May 6, 1913, by Dr. D. C. Perkins of Jamestown, N. Y.

Patient, Mrs. D., German, housewife, family history negative, mother of eight living children, the youngest five years of age. Patient had always been well up to about January 1, 1913, when she began to have an occasional attack of indigestion associated with vomiting, headache, coated tongue, abdominal pains and unusual constipation. Menstruation regular, piles, and occasionally noticed blood in stools. The above condition persisted, attacks re-occurring about every two weeks, until she entered The Jones General Hospital, Jamestown, N. Y., April 24, 1913, nearly four months after her first attack.

Patient's examination on entering hospital revealed a fairly well nourished woman, but who stated she had lost about fifteen pounds in weight in the previous three months. Physical examination negative, except coated tongue, slight tenderness over the whole abdomen and a few internal hemorrhoids. Temperature 99.5°, pulse 100, respiration 22, urine alkaline, specific gravity 10.18, no sugar, no albumin, complained of some abdominal cramps and did not care for diet.

During the twelve days following admission to hospital, the temperature varied between 99 and 100°, pulse between 90 and 120, respiration 20 to 25, daily vomiting spells, coated tongue, moderate abdominal pains, dark brown stools, and urine normal.

On the thirteenth day after admission, May 6, patient had a severe fainting spell at 10 A.M. after which complained of very severe pain in abdomen, vomited bile, pains continued until 7 P.M. when I was called in consultation. Patient very much under the influence of morphine, abdomen moderately distended and extremely tender, knees flexed, vomiting bile, temperature 99.4°, pulse 140, respiration 32. Diagnosis, obscure abdominal lesion. Patient was immediately prepared for operation.

Under ether anæsthesia by Dr. William M. Bemus, and assisted by Dr. Perkins, by median line incision, the abdomen was opened, in which there was about one pint of brownish fluid and about six feet of gangrenous small intestine extending from a point about four inches from the ileocæcal valve. The mesentery was markedly thickened and œdematous and in it could be seen and felt the thrombosed veins and arteries; all other abdominal organs normal except slight congestion. The fluid was sponged out and 72 inches of ileum resected; the incision at both ends of the diseased intestine being made through the healthy bowel about three or four inches from the line of demarcation. As much as possible of the diseased mesentery, containing the thrombosed vessels, was removed with the diseased bowel. Both ends of the normal bowel were closed and a lateral anastomosis made between lower part of remaining ileum and cæcum.

Punctured cul-de-sac and inserted rubber drainage tube into vagina. The intestines were gently cleansed with salt solution and the abdominal incision closed without drainage.

Patient received subcutaneous salt solution during the following 48 hours, followed by nutritive enemata for five days when they were substituted with beef juice by mouth.

During the three days following operation there was profuse discharge of brownish fluid through cul-de-sac drainage tube. The abdominal wound healed promptly, patient made an uneventful recovery and was discharged from the hospital well, four weeks after her operation. At the present writing, which is six months since her operation, patient is well and is able to do the household work for her family of seven.

Much effort has been made, without success, by Dr. Perkins and myself to discover the etiological factor in this particular case.

Jamestown, N. Y.

F. H. NICHOLS, M.D.

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